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SECTION OF MEDICINE

WEDNESDAY MORNING, JUNE 8, 1927

THE Section of Medicine of the Massachusetts Medical Society convened at nine-thirty o'clock in the ballroom of the Hotel Statler, Boston, Massachusetts, Dr. Hervey L. Smith, Springfield, presiding.

CHAIRMAN SMITH: I regret very much to announce that Dr. A. P. Merrill of Pittsfield, the Chairman of the Section, is not here. No word has come from him. Dr. Merrill never before missed a medical meeting in his life that I know of and I am very sorry that he is obliged to miss this one.

I have taken the liberty to ask Dr. Steele of Springfield to act as Secretary, and we will proceed at once to the papers.

The first paper, "What Can Be Learned from Urinary Examinations," is by Dr. William R. Ohler of Boston.

WHAT CAN BE LEARNED FROM URINARY EXAMINATIONS*

BY W. RICHARD OHLER, M.D.

My purpose is to limit the discussion of the subject to two general diseases, diabetes and nephritis. May I say at the start that I do not wish to detract in any way from the value of the more elaborate laboratory tests, such as the blood sugar and blood $C O_2$ in diabetes, and the blood nitrogen in nephritis. For the present, I consider that my job is to show just how much or how little can be learned from the simplest of laboratory tests—namely, urinary examinations.

I—DIABETES

It is assumed that every physician is familiar with two tests, (1) the qualitative test for sugar in the urine, and (2) the test for diacetic acid in the urine. Directions for performing these two tests are given below:

*Read in part before the meeting of the New Hampshire Surgical Club on May 3 under the title of "Practical Application of the Laboratory as It Relates to Urine Analysis and Blood Chemistry."

(1)

BENEDICT QUALITATIVE TEST FOR SUGAR IN URINE

Articles Needed

- (1) Benedict Qualitative Solution. (2) Medicine Dropper. (3) Test Tube. (4) Cup. (5) Water. (6) Heat.

The Test

- (1) Boil water one to two inches deep in a cup. This can be done over a gas stove, solid alcohol, electricity, or with an electric hot point put into the water.
- (2) Clean medicine dropper three times with water.
- (3) Put eight drops (no more, no less) of urine into a clean test tube.
- (4) Put one inch of Benedict Solution into the test tube on top of the urine.
- (5) Then put test tube containing urine and the Benedict Solution into the boiling water and leave there for exactly five minutes.
- (6) At the end of five minutes remove tube from boiling water.
- (7) Turn off heat.
There is no sugar present if the solution remains clear enough so that one may read through it.
- (8) The test tube should be emptied and cleaned promptly after the test is performed.
- (9) Clean the medicine dropper three times with water.

(2)

FERRIC CHLORIDE TEST FOR DIACETIC ACID IN URINE

Articles Needed

- (1) $\frac{1}{2}$ Watery Solution of Ferric Chloride any strength from 10 to 30%. (2) Medicine Dropper. (3) Two Test Tubes. (4) Cup. (5) Water. (6) Heat.

The Test

- (1) Put half an inch of urine into a test tube.
- (2) Add Ferric Chloride solution to the urine drop by drop until there is no cloud.
- (3) Keep shaking it so that the cloud will go away with the least possible amount of Ferric Chloride.
- (4) If the result has no reddish tint, there is no diacetic acid.
- (5) If there is any reddish tint to the result, pour half of the result into the other test tube and put it in boiling water for five minutes.
- (6) If at the end of this time the heated tube is less red than the one which was not heated, then there is diacetic acid present in the unheated urine.

*Tincture of Ferric Chloride will not serve.

I have mentioned only the qualitative test for urinary sugar, because one can get a pretty good idea of the percentage of sugar present by noting the color reaction from the Benedict test. For example, in the tubes passed around, the color reactions check as follows:

| | |
|----------------|---------------|
| Greenish color | 0.6 per cent. |
| Yellow-green | 1.2 per cent. |
| Brown | 2. per cent. |
| Red | 4. per cent. |

In handling insulin cases, if single urine specimens are examined (both for sugar and diacetic acid) before each insulin dose, then the color reactions give us a fair idea of the amount of insulin to use. For example:

| | |
|----------------|----------|
| Greenish color | 5 units |
| Yellow-green | 10 units |
| Brown | 12 units |
| Red | 15 units |
| Diacetic Acid | 15 units |

In other words, if single urine specimens are examined frequently, and if attention is paid to the color reactions and to the presence of diacetic acid, then in a general way the diabetic can be followed very satisfactorily by two simple urinary examinations—procedures which require nothing very elaborate in the way of laboratory equipment.

II—NEPHRITIS

During the past ten or fifteen years much has been written about various laboratory tests in nephritis. Many of the tests described have been very complicated, and moreover, many tests have been hailed as valuable only to be discarded later for some other test. All this has tended to shroud the whole subject in mystery; it has made it difficult to separate the practical from the impractical, and has given the impression that only specialists were qualified to handle the subject.

Recently the pendulum has been swinging in the other direction. We are beginning to appreciate just how much and how little even the most complicated tests will tell us. At present my purpose is to show how thorough a study can be made of nephritic problems with simple laboratory equipment and without special technical skill.

A. What can be learned about the diagnosis and prognosis of nephritis from urinary examinations alone?

For the purpose of this discussion it is assumed that by urinary examination we mean:

1. Amount.
2. Specific Gravity.
3. Test for Albumin.
4. Microscopic Examination of Sediment.

I appreciate that it may not be possible for the practitioner to do all of the above tests with any degree of regularity. Consequently, I shall confine my remarks largely to the first three:

amount, specific gravity and albumin, with the understanding that I am not detracting in any way from the value of routine examination of urinary sediments.

1.—Acute Nephritis

a. Diagnosis.

History very important.

Urine—small in amount, of high specific gravity and contains albumin, blood and casts.

b. Prognosis.

(1) Amount. It is very important to follow the urinary excretion chart. Even in cases where there is definite nitrogen retention in the blood, one can often anticipate a fall in blood nitrogen by noting a rise in urinary output.

(2) Specific Gravity. With improvement in urinary excretion, the specific gravity tends to fall. During convalescence, a tendency toward fixation of specific gravity at a low level suggests the possibility of chronic kidney damage. On the other hand, the persistence of a high specific gravity together with clinical signs, suggest the possibility of a sub-acute type of kidney damage.

(3) Albumin. Albumin may persist in small amounts long after the acute symptoms have subsided. Possibly this is of no great significance in the absence of other findings. Large amounts of albumin, especially intermittent attacks associated with clinical signs, small amounts of urine of high specific gravity, suggest the sub-acute type of lesion.

(4) Microscopic findings. The presence or absence of blood is of great importance. Especially should this be looked for after the patient is allowed up, because this finding may serve as a valuable test of kidney function.

2—Chronic Nephritis

a. Diagnosis. As we all know, the diagnosis of chronic nephritis is not always an easy matter. No laboratory procedures, whether simple or complex can take the place of a careful history and physical examination. Insofar as the routine urine examination is concerned (amount, specific gravity and albumin), occasional nocturia and persistent albuminuria, together with indefinite clinical symptoms may be the earliest sign of a beginning chronic nephritis. On the other hand, a single routine urine examination often gives entirely negative results. If simple routine examinations are to be of real value, they must be frequent and must include separate specimens as well as twenty-four hour amounts. Personally, I believe that the examination of a single office specimen is often of more value than the examination of a twenty-four hour amount.

Special Urinary Tests:

Certain special urinary tests are at our disposal which are but elaborations of a simple routine test, and which can be done without

much in the way of laboratory equipment. These are the so-called Two-hour Test and the Dilution-Concentration Test.

1—The Two-hour Test for Fixation of Specific Gravity

a. Principle. Normally fluids are excreted rapidly, solids slowly. In a given twenty-four hour period therefore, there will be considerable variation in amounts of urine and in specific gravity. Also, normally—unless considerable fluid is taken during the night—the night urine is relatively small in amount and high in specific gravity.

b. Method.

1. Meal hours:

| | |
|-------------------------|-----------|
| Breakfast | 8 |
| Dinner | 12 |
| Supper | 5:30 or 6 |
| Breakfast (next day) | 8 |

The heaviest meal to come in the middle of the day; absolutely nothing is to be taken between meals, not even water, and nothing from supper on the day of the test until breakfast the next day.

2. Diet:

Eat an ordinary hearty diet to include meat, salt, clear soup. The idea is to put the kidneys under a certain amount of strain.

3. Fluids:

Drink the equivalent of 8 to 10 tumblers, and use more fluids at dinner than at the other meals. By fluids is meant to include water, tea, coffee, milk, etc. Keep an account of the amount of fluids used.

Detail for Test Day

| | |
|---------|--|
| 8 A. M. | Pass urine. Need not be saved. |
| | Breakfast. |
| 10 | Pass urine. Save all in Bottle No. 1. |
| 12 Noon | Pass urine. Save all in Bottle No. 2. |
| | Dinner. |
| 2 P. M. | Pass urine. Save all in Bottle No. 3. |
| 4 | Pass urine. Save all in Bottle No. 4. |
| 5:30 | Supper. |
| 6 | Pass urine. Save all in Bottle No. 5. |
| 8 | Pass urine. Save all in Bottle No. 6. |
| 8 A. M. | Pass urine. Save all in Bottle No. 7. |
| | Breakfast. |
| | In Bottle No. 7 or No. 8 save all urine passed during the night. |

Note that the only urine to be thrown away is the first specimen on the day that the test is started. By saving the 8 A. M. specimen on the next day, the 24-hour period is completed.

c. Value. After this test is done, we have seven or eight single specimens of urine to be examined for amount, specific gravity, sugar, albumin, and microscopic elements.

Intrinsically, such a test is of much greater value than the routine examination of a single urine specimen or a 24-hour amount. For example, we are often able to detect the presence of sugar in some of the specimens, while in other specimens there is none; and we often find albumin and microscopic elements in some specimens and not in others. This suggests the importance of the examination of several single urine speci-

mens in the diagnosis of mild diabetes, obscure pyelitis and transient albuminuria.

Value in Nephritis—This test is of considerable value as an aid to the diagnosis of nephritis. The charts given below are from a few selected cases, with the idea of illustrating the value of the test in diagnosis.

1. NORMAL

| | | | | | | | |
|---------|-------------------------|--------|--------|-------|--------|-------|--------|
| Time | 8-10; | 10-12; | 12-2; | 2-4; | 4-6; | 6-8; | 8-8. |
| Amt. | 150cc; | 40cc; | 145cc; | 50cc; | 210cc; | 70cc; | 375cc. |
| Sp. Gr. | 1008; | 1028; | 1014; | 1027; | 1009; | 1025; | 1022. |
| Alb. | None. | | | | | | |
| Sugar | None. | | | | | | |
| Output | —1040cc; Intake—1800cc. | | | | | | |

Note (1) the wide variations in amounts of urine and in specific gravities; (2) that the specimens passed directly after meals are high in amount and low in specific gravity in contrast to the specimens passed between meals; (3) that the night urine is relatively small in amount and high in specific gravity.

2. EARLY DAMAGE

| | | | | | | | |
|---------|-------------------------|--------|--------|--------|-------|--------|--------|
| Time | 8-10; | 10-12; | 12-2; | 2-4; | 4-6; | 6-8; | 8-8. |
| Amt. | 160cc; | 200cc; | 75cc; | 125cc; | 60cc; | 105cc; | 725cc. |
| Sp. Gr. | 1016; | 1013; | 1017; | 1015; | 1019; | 1011; | 1014. |
| Alb. | 0 | S.P.T. | S.P.T. | S.P.T. | 0 | 0 | 0 |
| Sugar | None. | | | | | | |
| Output | —1450cc; Intake—2000cc. | | | | | | |

Note (1) that between the lowest and highest specific gravity there is considerable variation (8 points), but that there is a definite tendency toward fixation of specific gravity, if the test is studied as a whole; and (2) that the night urine is high in amount and low in specific gravity.

3. EARLY DAMAGE

| | | | | | | | |
|---------|-------------------------|--------|--------|--------|--------|-------|--------|
| Time | 8-10; | 10-12; | 12-2; | 2-4; | 4-6; | 6-8; | 8-8. |
| Amt. | 110cc; | 50cc; | 125cc; | 190cc; | 160cc; | 80cc; | 500cc. |
| Sp. Gr. | 1012; | 1018; | 1015; | 1010; | 1010; | 1014; | 1006. |
| Alb. | S.P.T. in each. | | | | | | |
| Sugar | None. | | | | | | |
| Output | —1215cc; Intake—2000cc. | | | | | | |

Note that this test gives results similar to the one above, except that after twelve noon, the specific gravities tend to be fixed at a lower level and the night specific gravity is very low.

4. EARLY DAMAGE

| | | | | | | | |
|---------|-------------------------|--------|--------|--------|--------|--------|--------|
| Time | 8-10; | 10-12; | 12-2; | 2-4; | 4-6; | 6-8; | 8-8. |
| Amt. | 320cc; | 250cc; | 280cc; | 210cc; | 200cc; | 270cc; | 235cc. |
| Sp. Gr. | 1010; | 1005; | 1004; | 1007; | 1011; | 1015; | 1025. |
| Alb. | S.P.T. in each. | | | | | | |
| Sugar | None. | | | | | | |
| Output | —1765cc; Intake—2000cc. | | | | | | |

Note that in this test there is a definite tendency toward fixation of specific gravity during the day, but the night urine is normal in both specific gravity and amount. This is from a proved case of chronic nephritis first seen when the diagnosis was in doubt. The test shows that the rather common practice of merely dividing the twenty-four hour amount into two twelve hour specimens, day and night, might give erroneous information.

5. MARKED DAMAGE

Time 8-10; 10-12; 12-2; 2-4; 4-6; 6-8; 8-8.
 Amt. 300cc; 190cc; 150cc; 235cc; 165cc; 320cc; 610cc.
 Sp. Gr. 1009; 1012; 1009; 1008; 1010; 1007; 1010.
 Alb. S.P.T. in each.
 Sugar None.
 Output—1970cc; Intake—2000cc.

Note that the specific gravity throughout the twenty-four hour period is pretty definitely fixed at a low level. In other words, the kidney has lost the ability to concentrate.

6. KIDNEY CONGESTION

Time 8-10; 10-12; 12-2; 2-4; 4-6; 6-8; 8-8.
 Amt. 30cc; 45cc; 25cc; 25cc; 65cc; 50cc; 300cc.
 Sp. Gr. 1026; 1026; 1026; 1026; 1022; 1022; 1022.
 Alb. Trace in each.
 Sugar None.
 Output—545cc; Intake—1580cc.

Note (1) that there is a tendency toward fixation in both amount and in specific gravity; (2) that the specific gravities are high and (3) that there is a poor response to fluid intake and a relative nocturia.

This sort of reaction is commonly found in kidney congestion, secondary to cardiac disease.

General Comment:—There are very few laboratory tests which tell us all we want to know and which are not subject to certain discrepancies. Concerning the test mentioned above, it is often necessary to repeat the procedure before interpretations can be made. In general, however, the two-hour test gives information which is of definite value as an aid to the diagnosis of chronic nephritis, especially in the chronic glomerular, interstitial or arteriosclerotic types.

In the type which is spoken of as nephrosis—or tubular—the test is of no great value, but here the clinical findings and routine urinary findings are sufficient to give a good idea of the condition, and more elaborate laboratory tests are often necessary to complete the diagnosis.

Finally, the two-hour test is of no great value as a help in prognosis, except that with advancement in kidney damage, the fixation of specific gravity becomes more complete and at a progressively lower level. Here also, more elaborate studies such as the blood urea nitrogen or non protein nitrogen, often throw additional light on the prognosis.

2—Dilution-Concentration Test

(Described by Pratt in the BOSTON MEDICAL AND SURGICAL JOURNAL of July, 1926.)

a. In principle, this test is practically the same as the Two-hour Test. The method of procedure is given below. For examples of normal and abnormal reactions, readers are referred to the original article.

b. Method.

Void urine at exactly 7 A. M. This specimen need not be saved. Immediately after, drink 1 quart (1000cc.) water or part water and part tea.

7:30 or 8. Patient may eat rolls, toast and an egg, or any other dry food.

| | | |
|---------|---|---|
| 8 A. M. | exactly, urine voided and all saved in Bottle 1 | 1 |
| 9 | " " " " " " " " | 2 |
| 11 | " " " " " " " " | 3 |
| 1 P. M. | " " " " " " " " | 4 |
| 3 | " " " " " " " " | 5 |
| 5 | " " " " " " " " | 6 |
| 7 | " " " " " " " " | 7 |
| 9 | " " " " " " " " | 8 |

Between 9 P. M. and 7 A. M. next day, collect all urine in Bottle No. 9, the last voiding to be at exactly 7 A. M.

No fluids to be taken after 7 A. M. until 7 P. M. or 9 P. M., and then the amount is to be measured. Relatively dry food is given at lunch and supper, such as potato, cheese, eggs, meat and toast.

In the Two-hour Test and the Dilution-Concentration Test, we have two valuable methods for the study of nephritic patients. Both tests are simple, they can be done at home by patients of merely average intelligence, and for the physician they require the minimum amount of laboratory equipment.

In addition to remarks on diabetes and nephritis, something should be said about periodic urine examinations. It is apparent that the public is willing to have urine examinations made, and willing to pay for such work. In my opinion the physician is the one individual to take charge of this matter. If he cannot actually perform the tests, he should at least interpret the findings to the patient. Too often the whole matter is left to some commercial laboratory.

If it is a question of the diagnosis of nephritis, surely the entire responsibility of arriving at a correct diagnosis rests with the attending physician. On the other hand, there are many perfectly well individuals past middle life, who show a little albumin in the urine. Often these individuals need to be protected from the false interpretations given such findings by commercial laboratories.

Finally, no case of infection, no matter how slight, is well treated unless treatment includes a urine examination. The lack of such a routine practice means that cases of acute nephritis and pyelitis are frequently missed.

In answer then to the question, "What can be learned from urinary examinations?"—much can be learned from examinations which include only amount, specific gravity, albumin, sugar, diacetic acid, and much more from examinations which include a microscopic study. However, it is one thing to talk about urine examinations, and quite another thing to get them done. Herein lies the crux of the whole matter.

In conclusion, let me say that the few mistakes I have seen in medicine have arisen not because the attending physician failed to ask for some complicated test like a basal metabolism test or a blood nitrogen, but because he failed to do a simple routine urine examination.

CHAIRMAN SMITH: At this time it is necessary to appoint a nominating committee for the officers of the Section next year. I will appoint on that committee Dr. A. A. Hornor of Boston, Dr. W. B. Breed of Boston and Dr. M. W. Pearson of Ware. They will bring in a candidate for chairman and secretary for next year.

The next paper, "The Diagnosis of Renal and Ureteral Lesions from the Viewpoint of the Genito-Urinary Surgeon," will be read by Dr. George Gilbert Smith of Boston.

THE DIAGNOSIS OF RENAL AND URETERAL LESIONS FROM THE VIEWPOINT OF THE GENITO-URINARY SURGEON

BY GEORGE GILBERT SMITH, M.D., F.A.C.S.

ON June 9th, 1893, thirty-four years ago almost to a day, James Brown of Johns Hopkins University succeeded in catheterizing through a cystoscope the ureter in a male. Brenner, five years before, had been the first to catheterize the ureter in a woman. (*Young's Practice of Urology*, Vol. II, p. 245.) These early attempts to study the upper urinary tract were carried out by instruments which today would seem to us as awkward and inefficient as instruments of a prehistoric civilization. In 1897 Albarran devised the hinged lever as a means of guiding the catheter into the ureter; since then this principle has been employed in all indirect vision cystoscopes.

The early years of the 20th Century saw much improvement in the finer details of the cystoscope. Excellent instruments were manufactured by American firms and the cystoscope came to be generally used as a means of investigating diseases of the urinary tract. Before that time, genito-urinary surgery consisted in catheterizing overdistended bladders, crushing vesical calculi, and operating upon fairly obvious lesions. With the perfection of the cystoscope and the development of the X-ray, what had before been a matter of conjecture became susceptible of actual demonstration. Today it is quite unusual to find a pathological condition of kidney or ureter which cannot be diagnosed with a fair degree of exactness. Mistakes in diagnosis are still made and probably always will be, but they are almost always due to errors in observation, to failure to determine all the essential data, or to incorrect interpretation of those obtained.

Modern methods of urological diagnosis demand a wide knowledge of medicine and surgery. The "specialist" who views a urological problem solely from its urinary aspect cannot succeed in his work. The differential diagnosis between lesions of kidney and ureter and disease of the intraperitoneal organs requires as much consid-

eration of the latter as of the first. More and more we are realizing the relationship between kidney infections and foci elsewhere in the body, and that between persistent pyelitis and ptosis and intestinal indigestion. The dependence of satisfactory renal function upon a vigorous heart muscle and a sufficient blood pressure is recognized. To appreciate these and other factors in the patient's condition, the genito-urinary surgeon must take a careful history and do a complete physical examination. Naturally he is interested in palpation of the kidneys. When this is done, not by means of a few rough jabs beneath the ribs, but by gentle, attentive bimanual pressure while the patient inhales deeply, much information may be obtained. Upon palpating a kidney recently, I felt a grating sensation as if stones were being rubbed together. Several internes who were present got the same sensation, and we made a diagnosis of renal calculi which was later confirmed by the X-ray.

Examination of the urine for its bacterial as well as its chemical and microscopic features is obviously an important step in the study of a case; if the patient is a female, the specimen should be obtained by catheter. X-ray of the urinary tract is desirable in practically every case of suspected kidney or ureteral disease. A good X-ray should show the outline of the kidneys, from which we may learn whether they are normal in size and position, and regular in contour. It should show the shadows of stones. Nowadays very few calculi escape detection, although those consisting of cystin, xanthin, fibrin, cholestrin or leucin (*Young's Practice of Urology*, Vol. I, p. 374) may throw a shadow so slight as to be almost invisible.

These investigations form the foundation of a urological diagnosis; in some instances they yield only negative results, and further investigation by means of the cystoscope and the ureteral catheter is necessary before a diagnosis can be made. In other cases the diagnosis may be arrived at by the preliminary tests alone, but a complete urological diagnosis must include many more facts than are learned from these simpler tests. For example, the X-ray may show in the kidney region a shadow which is interpreted as that of a calculus. This diagnosis may be made more certain by the presence of blood or pus in the urine. Yet the genito-urinary surgeon who has to decide what should be done about this condition must know many more facts than these. He must know, not only that there is a stone in the kidney, but to what extent the stone has damaged the kidney. These facts may be learned by pyelography, which will show the relation of the stone to the renal pelvis, and by functional tests. The surgeon must also know the condition of the other kidney, so that at operation his judgment as to whether the kidney should be removed or not, may rest upon already ascertained facts.

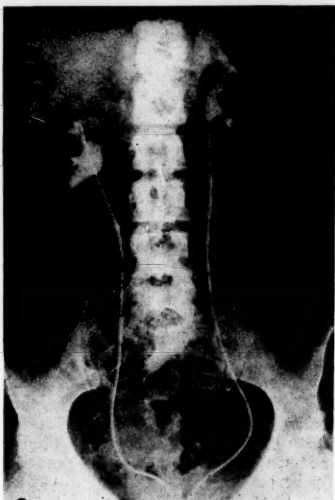


FIGURE 1. Left kidney pelvis normal. Right kidney pelvis shows evidence of slight obstruction at uretero-pelvic junction quite possibly due to an aberrant vessel. Urine uninfected. An appendectomy was done and treatment of general ptosis was instituted. Patient was in much better health two years after these studies were made.

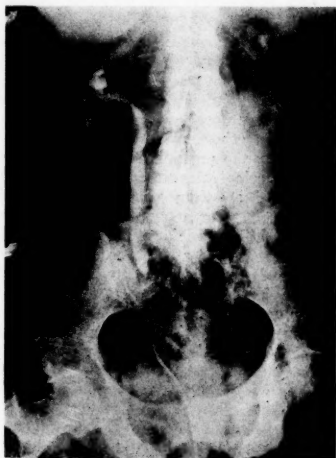


FIGURE 2. Stricture of ureter at pelvic brim. Three years previously the patient had been treated with radium because of carcinoma of the uterus. The stricture may have been caused by constriction by a metastasis.



FIGURE 3. Hydronephrosis of an ectopic or pelvic kidney.



FIGURE 4. Cystogram of a boy 8 years of age, with regurgitation up both ureters. Following an operation for spina bifida done 18 hours after birth, the nerve supply of the bladder was damaged, leaving the boy with urinary incontinence and a residual urine of 4 to 6 ounces. The ureters are dilated, but the kidney pelves show only slight changes. The renal function was unimpaired.

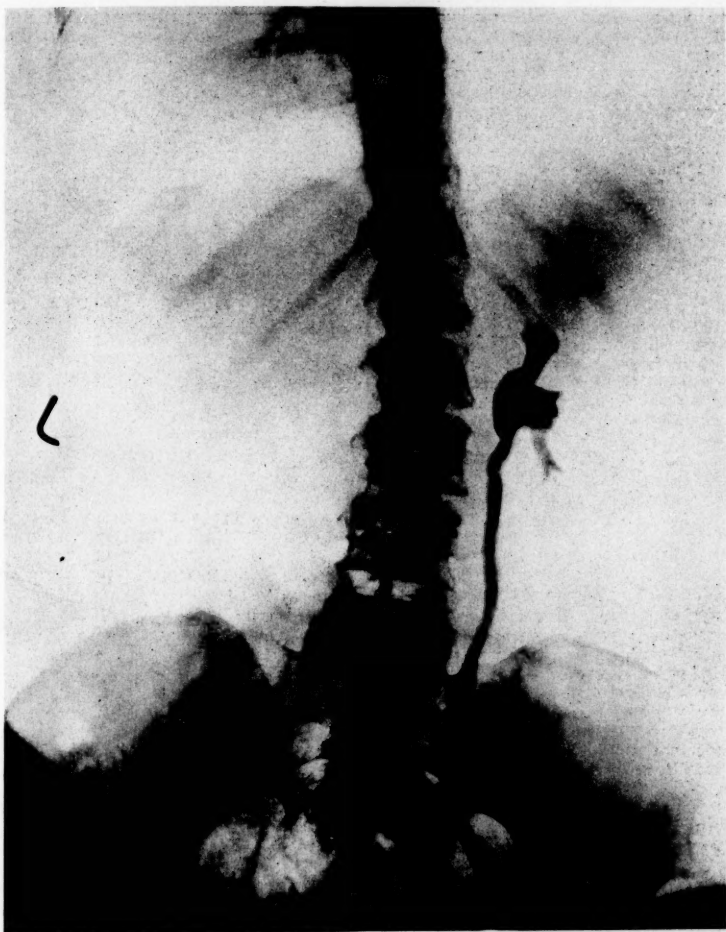


FIGURE 5. Recurrent pyelitis in a woman of 50. The right kidney pelvis shows very slight dilatation, and at the uretero-pelvic junction a suggestion of constriction as by an aberrant vessel.

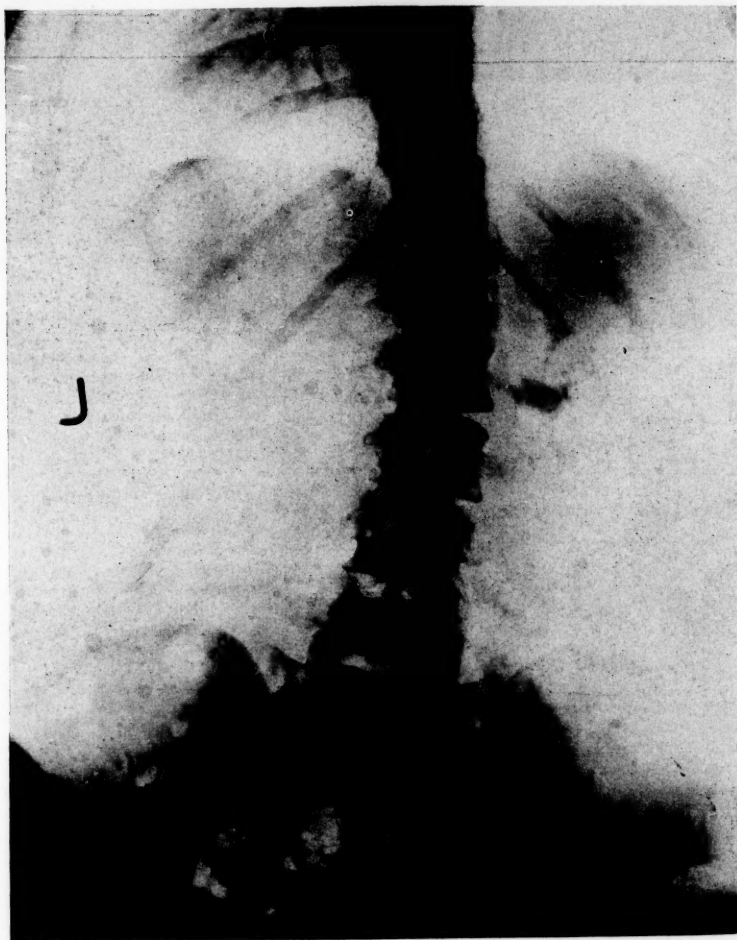


FIGURE 6. The same kidney as shown in the preceding plate, 6 minutes after the pyelogram was taken. The pelvis still retains some of the sodium iodide solution; the emptying time is slightly delayed.

By means of pyelography, which was introduced about 1914, great refinement of diagnosis of renal and ureteral lesions has been made possible. Examples of this can best be illustrated by concrete cases, and will be shown in lantern slides later. There are, however, some types of renal and ureteral disease in which even the most careful cystoscopic study will not yield positive information. The diagnosis then de-

casts. In a third type, the diagnosis of which depends upon circumstantial evidence alone, the ureter of the affected side is so distorted or strictured that catheterization is impossible. This sort of case is found most frequently as a result of renal tuberculosis. If the stricture completely closes the ureter, a shut-off pyonephrosis occurs, and the urine obtained from the bladder may be perfectly normal. Cystoscopy usually



FIGURE 7. A totally destroyed hydronephrotic left kidney, removed at operation.

pends upon the exclusion of other possibilities, upon circumstantial evidence, and upon the surgeon's experience and reasoning powers. Prominent among these cases are those of perirenal infection. The urine from the kidney in question may be normal or only slightly abnormal; the function and pyelogram may not indicate disease. The surgeon must make his diagnosis by the exclusion of other lesions, by the evidences of infection, such as fever and leucocytosis, and by the presence of a tender mass in the renal area. Another type of case is that of so-called essential hematuria. One excludes renal tumor by the normal pyelogram and function and stone by the negative X-ray. Tuberculosis and pyelitis are excluded by the absence of pus and bacteria in the urine. Chronic nephritis is excluded by normal blood chemistry, specific gravity fixation tests and the absence of

gives evidence of this condition through the changes in configuration of the ureteral meatus. The lack of function of the affected kidney may be demonstrated by the injection of indigo carmine intravenously, after which a jet of blue may be seen to issue from the ureter of the healthy kidney while there is no such jet from the diseased side. If phenolsulphonephthalein is used to estimate the renal function, the output of the dye from the healthy kidney may be secured through a catheter in the ureter, while the output from the uncatheterized ureter is obtained through a catheter placed in the bladder.

In many respects urological diagnosis has become a matter of manipulative skill, but more than the mere collection of facts is necessary. Like other laboratory findings, they cannot be taken at their face value alone, but must be critically checked and carefully interpreted.



FIGURE 8. The right kidney of the patient whose left kidney is shown in Figure 7. Marked hydronephrosis, due to an aberrant vessel. The ureter was transplanted to another portion of the pelvis after being carried to the other side of the aberrant artery. The redundant pelvis was cut down and resutured, and drained by a tube passed through the renal cortex.



FIGURE 9. The same kidney as shown in Figure 8, two years after operation. Although the pelvis is still large, the calices are more distinct, and drainage from the kidney is evidently improved. The patient has no symptoms referable to the kidney.

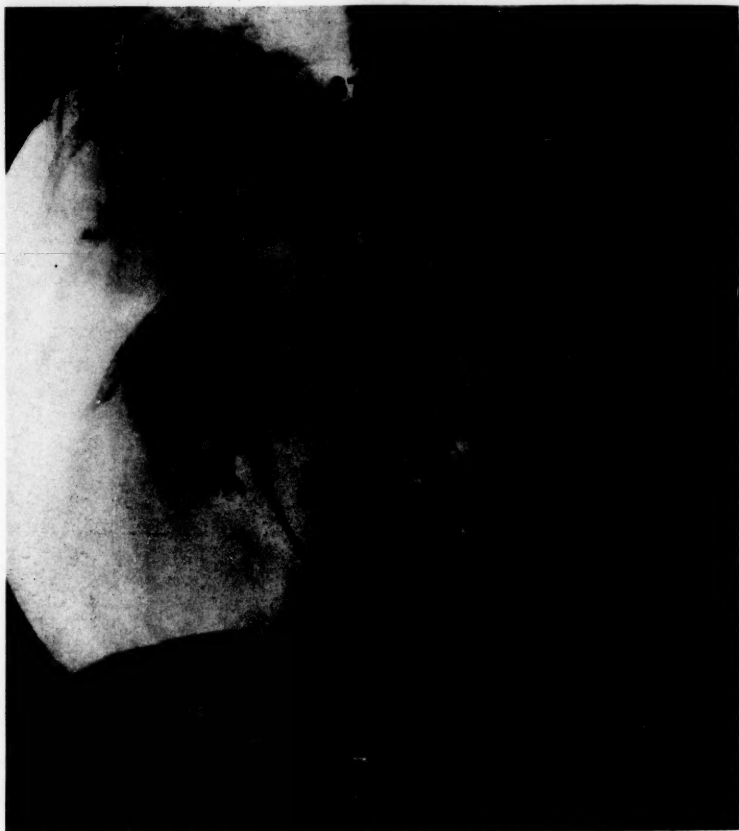


FIGURE 10. Pyelogram of the left kidney in a woman of 52. She was thought to have a simple pyelonephritis until this pyelogram was made. The round shadow lying to the outer side of the pelvis represented a thick walled, dilated calyx which communicated by a narrow orifice with the upper branch of a bifid pelvis. Nephrectomy was done.

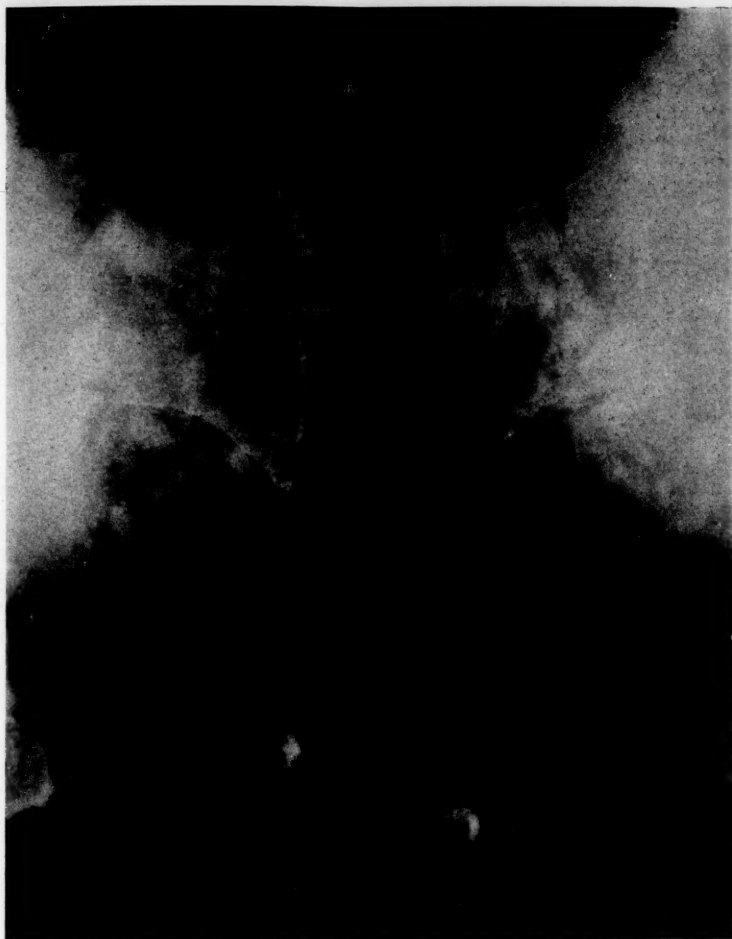


FIGURE 11. Y shaped ureter and double pelvis. The patient's complaint was hematuria. As the patient left Boston, later developments are not known.

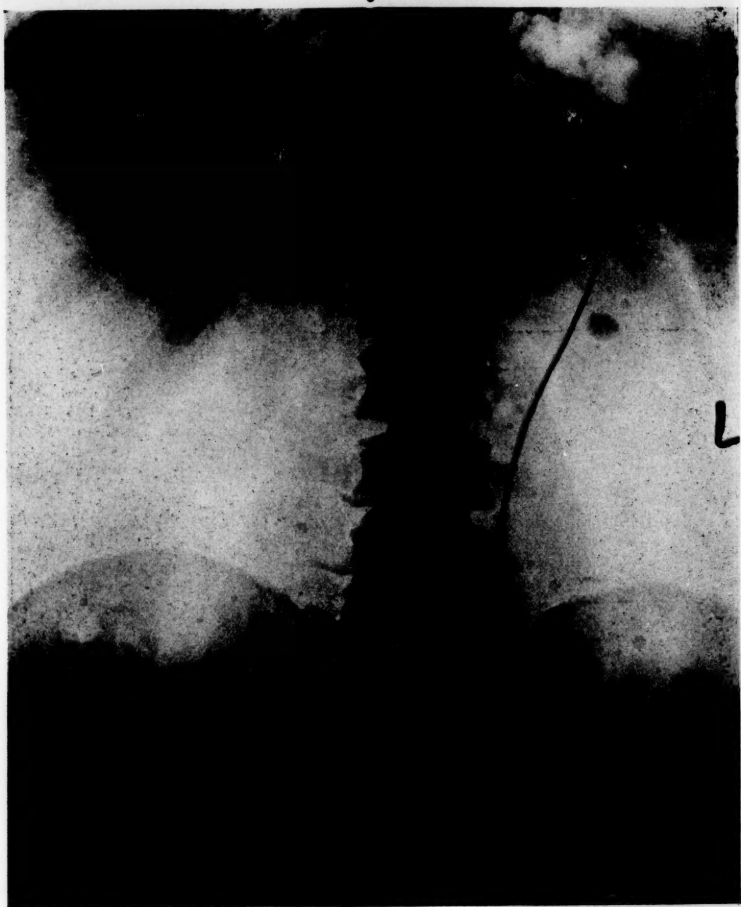


FIGURE 12. A stone in the renal pelvis.

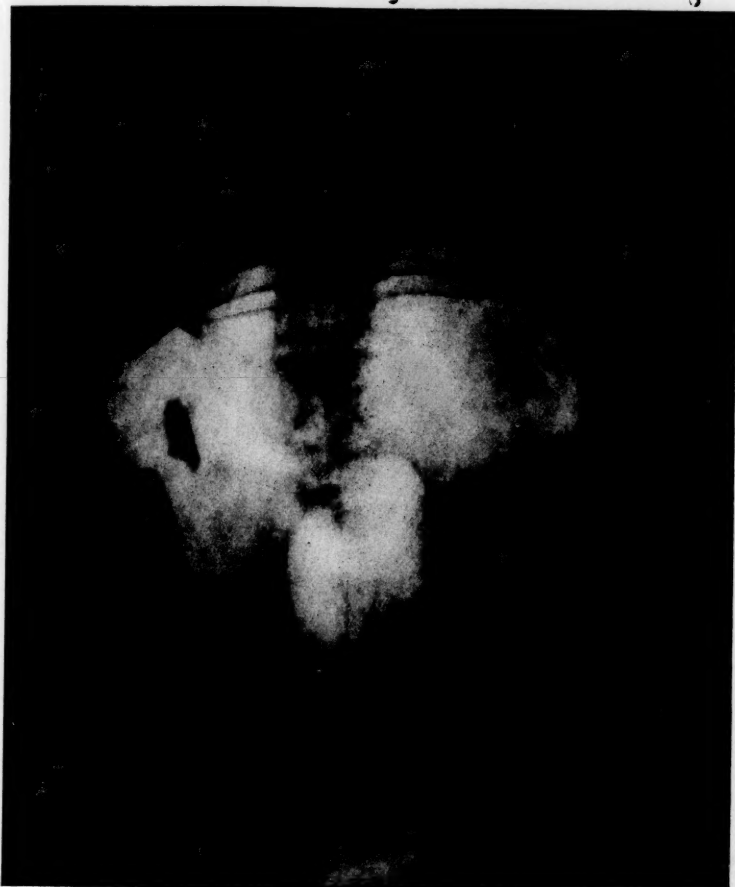


FIGURE 13. The same stone passing down the ureter.



FIGURE 14. The same stone lying in the bladder, where it was crushed and evacuated. The ureteral orifice appeared to be normal in size. The stone was oval in one plane, but quite flat in the opposite plane, and was smooth.

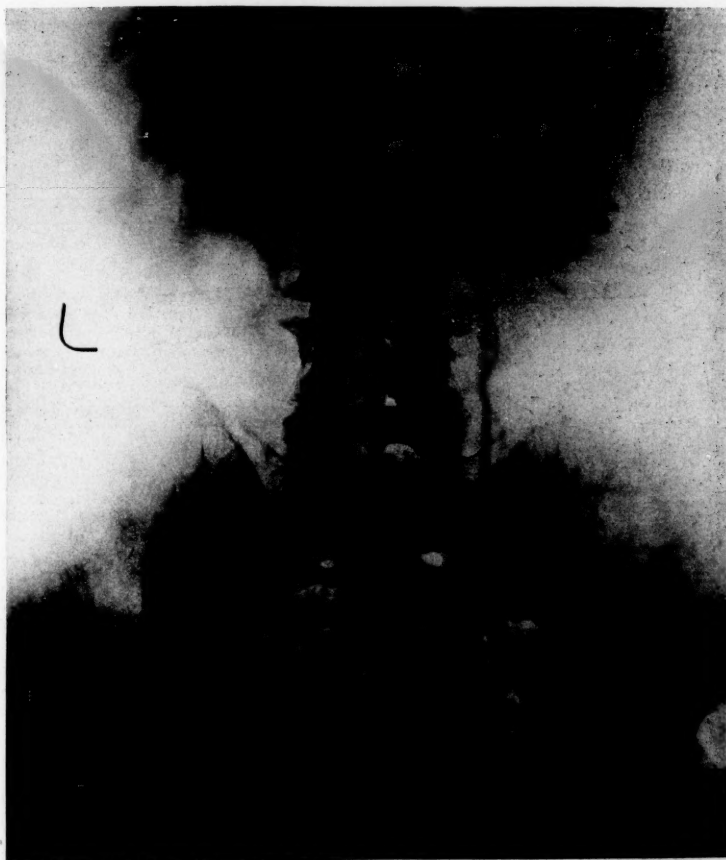


FIGURE 15. Pyelogram of right kidney in a man of 68. The distortion and obliteration of the pelvis is characteristic of renal tumor. Nephrectomy was done in March, 1925, and the hypernephroma shown in Figure 17 was removed. The patient was well two years later.



FIGURE 16. Another pyelogram of a kidney tumor in a colored girl 28 years of age. The growth was a rapidly growing carcinoma. The specimen is shown in Figure 18. Although there were metastases in the juxta aortic glands the kidney was removed, but the patient died two days after operation.

Not infrequently two sets of data may be obtained which appear to be diametrically opposed; the explanation of the discrepancy must be found. The wise surgeon will not depend upon one fact alone; he will want to back it up by substantiating testimony, and by obtaining such overwhelming proof of the existence of the suspected lesion that he can proceed with confidence to act upon the conclusion thus reached.

CONCLUSIONS

1. Modern methods of urological diagnosis, if properly applied, will usually supply sufficient data to allow one to arrive at a correct diagnosis.

2. These data, like other laboratory findings, must be interpreted and carefully checked; they cannot always be accepted at their face value.

3. Even with the employment of all methods of diagnosis, a certain small percentage of cases, notably those with inflammatory conditions outside the kidney and the so-called essential hematurias, are not susceptible of exact diagnosis.

4. Successful management of surgical lesions of the upper urinary tract requires not only that adroitness in technic which will enable the cystoscopist to ascertain the facts in a difficult case, but a knowledge of human physiology and pathology, and that combination of common sense, experience and imagination which constitutes "surgical judgment."



FIGURE 17. Drawing of a hypernephroma, the pyelogram of which is shown in Figure 15.



FIGURE 18. Drawing of a carcinoma of the kidney, the pyelogram of which is shown in Figure 16.

CHAIRMAN SMITH: The next paper, "The Clinical Value of Kidney Functional Tests," is by Dr. Henry C. Bugbee of New York.

THE CLINICAL VALUE OF KIDNEY FUNCTIONAL TESTS

BY HENRY C. BUGBEE, M.D., F.A.C.S.

THE accurate study of renal function made possible by laboratory tests of comparatively recent development has gone hand in hand with improved diagnostic procedures, the two placing urology on a thoroughly scientific basis.

It is not my purpose to question as to whether cystoscopy and urography, or kidney functional test, have contributed most toward the advancement of urological surgery, but to demonstrate through clinical observations the value of kidney functional tests in conjunction with physical signs and the specific study of the urinary tract, in arriving at a diagnosis, estimating the prognosis and outlining the treatment in individual cases.

If one assumes the attitude that laboratory tests supplement but do not replace direct observation of the patient; that the additional information gained from laboratory tests is indispensable; that no single laboratory test is conclusive, but that these tests are merely more evidence gained in completing the clinical picture we are trying to construct of the patient, their true value will be understood.

In recent years, the specific estimation of re-

nal function has in the hands of the urologist simmered down to the accurate data obtained through the study of blood chemistry and phenolsulphonephthalein elimination. Some have shown a tendency to rely upon one to the exclusion of the other, but the two tests, the one showing the blood retention and the other the true excretory capacity of the kidneys, are of inestimable value when employed together.

It is our routine in any given urological case in which we wish to ascertain the kidney function, to obtain a full blood chemistry (including blood urea, uric acid, creatinin, C O_2 combining power and blood sugar) at the same time estimating the phenolsulphonephthalein output.

Future tests consist of a repetition of blood urea estimations and the phenolsulphonephthalein output. Creatinin and uric acid estimations are also repeated if the patient is not improving, and blood sugar is taken at intervals if found to be high.

Probably the most important and striking class of cases presented for our study of renal function is that of prostatic obstruction, the back pressure incident to the increased effort to empty the bladder causing at first congestion of the kidneys and later true impairment of renal function. In these cases we are concerned with the combined function of the two kidneys, and here these tests are of decided value.

While in these cases as pointed out by Frontz and Geraghty¹ the estimation of blood urea is of value as an index to the amount of retention in the blood of nitrogenous end products, one must take into consideration the fact that the normal urea content of the blood is subject to considerable variation, recognizing a content of 20 to 40 mg to 100 cc of blood as being within normal limits and that 50 mg cannot be considered high in persons on a high nitrogenous diet; while on the other hand a blood urea of 30 to 80 mg in the presence of impaired function may on a restricted diet come down to a normal level only to rise on resumption of a nitrogenous diet. Protein intake, therefore, must be taken into consideration.

The amount of fluid intake is another important element entering into our final acceptance of the value of blood urea estimation, a marked decrease always following the ingestion of large amounts of water. As it is our custom to give our prostatic cases all the water it is possible for them to take, this becomes an important factor.

On the other hand, diet and fluid intake do not have to be considered in estimating the phenolsulphonephthalein output as the amount excreted is not influenced by either.

The following case illustrates the above points:

A man 74 years of age with a history of increasing difficulty of urination over a period of 10 years with several attacks of retention, presented a moderate

hypertrophy of the prostate and 8 ounces of residual urine. Under indwelling catheter drainage he was comfortable and showed no clinical manifestations of uremia. Although his tongue was never moist (the one best physical sign of renal insufficiency) his blood chemistry was normal (he being on a limited diet and large fluid intake); the phenolsulphonephthalein output, however, was low—never rising above 23% for two hours. Following a suprapubic cystotomy as the first stage of a prostatectomy, this patient had complete suppression for three days, resuming kidney function only after a blood transfusion. Even during the period of suppression the blood chemistry was normal, as he was taking little nourishment. During three months of suprapubic drainage the blood picture remained normal; the phenolsulphonephthalein output, however, slowly increased until it became stationary at 43% for two hours. Prostatectomy in this case was followed by no diminution in kidney function and his recovery was rapid.

This case is typical of many.

The quantitative determination of creatinin, an end product of endogenous metabolism, would seem theoretically to be an ideal means of estimating the degree of renal insufficiency, the amount of creatinin in the blood not being influenced as is urea by protein intake. However, a marked diminution in renal function can occur before there is any rise in the creatinin level. The normal blood content of creatinin averages about 2 mg per 100 cc and as a rule there is no appreciable increase in this amount until well-marked renal insufficiency is present, as determined by a high blood urea and low phenolsulphonephthalein output. An increase in creatinin to 5 mg or more in the presence of a high blood urea offers a grave prognosis.

The estimation of uric acid content of the blood is also of corroborative value. While the increase takes place more rapidly than does creatinin in kidney insufficiency, it is not more accurate.

The total estimation of function by means of phenolsulphonephthalein elimination will give us accurately the amount of reduction in the excretory capacity of the kidneys but does not tell us whether the excretory capacity is sufficient to meet the requirements of elimination in the individual case.

Our observations corroborate those of Braasch and Kendall² in that we have noticed in certain cases differences between the results following intravenous and intramuscular injections of phenolsulphonephthalein in the same individual. This difference has run as high as 20%, which has been interpreted as an indication of retention of waste products in the tissues themselves. This retention is further increased by acidosis.

For routine use we prefer the intravenous method as more accurate and the appearance time is shorter. Water should be given freely before and during the test to dilute the urine, diminishing the possibility of error from the retention of small amounts in the bladder. A catheter is used in cases of retention.

The investigations of Shaw³ of the elimination of phenolsulphonephthalein in normal and

abnormal conditions, giving a definite normal curve and significant variations from this in various diseases has proved of real value. Shaw suggested the collection of the urine at 15 minute intervals for 2 hours after the appearance time. The normal curve was characterized by an average output of 40% during the first 15 minute period, 17% during the second, 8% during the third, 4% during the fourth, with a gradual decrease to .5% during the eighth 15 minute period. In all the cases of known renal disease in which this test was applied, there were definite abnormalities in this curve and in several cases the presence of an abnormal curve indicated impending renal failure, while the other tests were negative.

The majority of the cases studied by Shaw were those of renal damage from back pressure and infection. In this group a striking feature was an increase in appearance time and a delay in the peak of elimination. In the cases followed with repeated tests, the peak of elimination occurred earlier as the kidneys improved while the two hours' output often remained unchanged.

The accuracy of these findings has been proven to me by observations in recent cases of prostatic obstruction with back pressure; however, half-hour periods of collection have been found satisfactory as a routine. A man of 84 showed a two hour output of phenolsulphonephthalein within normal limits—the peak of the elimination was but slightly delayed. After suprapubic drainage the figures for the first and second hours were reversed. Definite uremic symptoms were present, yet, with a stabilization of kidney function with again a reversal of the curve of phenolsulphonephthalein elimination approaching the normal, the uremic symptoms disappeared and he went on through his second operation to a recovery with no further clinical manifestations of renal insufficiency.

A case now under treatment gave only a trace of phenolsulphonephthalein during the first hour and 8% return during the second, a blood urea of 82 mg, yet under prolonged catheter drainage during which period he showed all the clinical signs of marked renal insufficiency the phenolsulphonephthalein curve rose to a peak of 12% in the second 15 minute period with a total of 35% for the two hours, and a blood urea of 23.5 mg. In this case suprapubic drainage and secondary prostatectomy have been followed by no reaction or uremic symptoms.

One could go on citing case after case of back pressure upon the kidneys resulting in renal insufficiency, such cases showing uremic symptoms at once with the break in renal balance resulting from relief of the back pressure—the dry coated tongue, malaise, loss of appetite, abdominal distention, lowered blood pressure, and dim-

inished urinary output being characteristic, these clinical signs being invariably accompanied by a lowered phenolsulphonephthalein output together in many instances with urea retention as shown by the blood chemistry.

As already stated, the clinical signs and symptoms fit in with the tests of function, the whole forming a characteristic picture, the tests being of great value in giving us an accurate working basis.

This study of renal function in cases of retention due to prostatic obstruction has convinced me not only of the value but necessity of slowly stabilizing these patients when renal balance has been broken. This can only be done by slowly relieving the back pressure and carrying out the operative work in stages. The resulting low mortality justifies this procedure. There is no more striking picture than that presented by these men of advanced years who are brought back to health, using these accurate data as a guide, a one time kidney function that would not seem to support life later becoming fixed at a certain level which although often much below normal is sufficient to meet the requirements of elimination in the individual.

The finding of an increase of blood urea and other substances considered can be taken as an indication of bilateral renal disease yet gives no information regarding the presence of a unilateral functional renal disturbance. The same can be said of the estimation of the combined phenolsulphonephthalein output; however, in the study of function of the separate kidneys by means of ureteral catheterization, noting the time of appearance and obtaining the total output of phenolsulphonephthalein by each kidney simultaneously, data of great value are obtained.

In the exceptional case in which the ureters cannot be catheterized, by filling the bladder with an alkaline solution and observing the ureteral orifices, the appearance time of the phenolsulphonephthalein in the urine from each kidney may be ascertained as well as the intensity of the color, in a manner similar to the technique employed after the intravenous administration of indigo-carmin. A urea estimation of the separate urines, also taken simultaneously, gives additional information regarding the relative function of the two kidneys.

From the standpoint of diagnosis such facts are of decided value, for while the chemical, microscopical and bacteriological examinations of the separate urines, in conjunction with the study of the symptoms and physical signs of a renal lesion, especially when verified by urography, may make the diagnosis positive, yet there is an occasional case in which clinical manifestations are absent, pyelograms negative or misleading, and in such instances the blood picture with separate kidney functional tests may lead us on to further study which results in a

definite diagnosis of the true pathology present.

I can recall cases of polycystic disease of the kidneys, also of early renal tumor, that have been diagnosed through the information obtained through renal functional tests. On the other hand the kidney has often been excluded in diagnosing an abdominal tumor through finding a normal renal function.

The differentiation of a pelvic from a parenchymatous infection of the kidney may be possible through a study of the function of the kidney in question.

Repeated functional tests are valuable in estimating the prognosis in renal lesions, in outlining treatment as well as noting its progress. Repetition also diminishes the possibility of technical errors.

When we come to the consideration of renal surgery we reach the peak in the value of functional kidney tests. Is the lesion unilateral or bilateral? What is the relative function of each kidney? Does the pathology present require surgical intervention for its relief? Is the function of the kidney preserved to a degree that justifies an attempt to save the kidney?—or will only a nephrectomy give promise of a cure? If a nephrectomy is indicated as the operation of choice or necessity, will its fellow meet the requirements of elimination in the individual?

How can these questions be answered except by a most thorough and often prolonged study of the function of the separate kidneys?

These studies have led to a more conservative position by the urologist regarding kidney surgery, especially in the management of cases of renal infection and of lithiasis. Kidneys that at one time were condemned have been found, by means of repeated tests, to regain function through treatment or conservative operative measures and a useful organ is often saved which formerly would have been removed at once. Operative results in renal tuberculosis, so often bilateral, the early diagnosis and thus the possible cure of renal tumor, must be partly attributed to our knowledge and study of renal function.

Thus we have tests of renal function that coincide with our clinical observations, cystoscopic and urographic studies, giving us data which (with full knowledge of their limitations) complete the picture and should be utilized at all times in diagnosing, outlining prognosis and treatment as well as checking off the progress of the treatment of urological lesions.

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- 2 William F. Braunsch and Kendall: *Jour. of Urol.*, 1921, V, 127-132.
- 3 E. C. Shaw: A Study of the Curve of Elimination of Phenol-sulphonaphthalein by the Normal and Diseased Kidneys. *Jour. of Urol.*, 13: 575-597, June, 1925.

CHAIRMAN SMITH: The Nominating Committee offers for your consideration for Chairman of next year's meeting Dr. William R. Ohler of Boston, and Secretary, Dr. Albert E. Parkhurst of Beverly.

If there are no other nominations, I will ask those in favor to say "aye"; those opposed. It is a vote.

The next paper is "Diuretics, Their Utility and Limitations," by Dr. Henry A. Christian of Boston.

DIURETICS, THEIR UTILITY AND LIMITATIONS

BY HENRY A. CHRISTIAN, M.D.

THE diuretic group of drugs is an important one in practical therapeutics. Used correctly they often produce striking improvement in our patients. In last analysis what they accomplish is an increased urinary output, which means an increased elimination of water, and dissolved in it various constituents of the circulating blood soluble in water and capable of elimination through the kidney. If the water intake of the body is decreased by limiting the ingested fluid below the level of urine output, then diuretics, if effective, cause a positive loss of water from the body. In large measure this output is from water stored in the tissue, for any water depletion of the circulating blood is quickly restored from the tissues. In addition, depletion of other substances must occur, though probably to a less extent. However, much less is known about the accelerated excretion of the normal urinary constituents other than water. Still less is known as to possible increased elimination of abnormal substances, when there is an increased elimination of water, though the assumption that this happens is the basis for increasing urinary output in various toxic conditions.

Today I am going to consider merely the water elimination of the kidney as influenced by diuretics, since that is easily measurable. At the same time it is certain that some increased elimination of other substances always occurs. The effect of diuretics on water elimination can be measured by comparing urine amount with fluid intake during twenty-four hour periods, and by weighing the patient at frequent intervals, the loss in weight over short periods of time being due, in the main, to water loss. It is best to make both observations on our patients, for at times the loss of water as indicated by decreased weight seems out of proportion to the increase in output of urine.

At present we have available several excellent diuretics, digitalis, caffeine, theophyllin or theocin, theobromin sodiosalicylate or diuretin, theophyllinaethyldiamin or euphyllin, and merbaphen or novasurol.

As therapeutic agents these various substances are used to remove fluid accumulated in

the body in such abnormal amounts as to cause uncomfortable symptoms or injurious reactions and to remove other substances which may exert a deleterious influence. The evidence of the former effect is obtained easily by measuring the urine and weighing the patient; that of the latter is more difficult to get, involving either chemical analysis of excreted substances, or in case of toxic substances of unknown nature, may be judged of only by observing improvement in the general condition of the patient. As indicated above, I will confine my remarks today to water elimination.

So far as water is concerned, diuretics are not needed unless the accumulated fluid, whether in the form of subcutaneous oedema or as ascites or hydrothorax, is causing symptoms; there seems to be no reason to attempt to remove small accumulations of fluid which cause no discomfort; or to put it another way, diuretics are not indicated unless oedema is excessive or much fluid has accumulated in body cavities. Consequently I will give no time to a discussion of the effect of these drugs except in patients definitely water-logged. As a rule, fluid in the thorax is not much affected by diuretics and should be removed mechanically by trocar whenever it embarrasses respiration.

It is to be remembered that, if a diuretic is effective, it increases the amount of work done by the kidney and this in itself may react injuriously on a seriously damaged kidney. Again it should be kept in mind that the kidney is an extremely vascular organ, which functions efficiently only when its circulation is efficient. Directly or indirectly a diuretic to increase urine flow appreciably must increase renal circulation to a very considerable degree. In other words, for good diuresis the kidney structure must be reasonably intact and the general circulation capable of providing an increased blood flow to the kidney. Very severe lesions of kidney or heart preclude any extensive diuretic response; by treatment heart work is capable of much increase, even though the heart is structurally badly damaged, and often this should be brought about by proper cardiac therapy before any special diuretic is used. In this sense digitalis often is a very perfect diuretic for patients with cardiac edema and no other is needed. Frequently, however, to obtain best results after digitalis is given to this type of patient for a few days an additional diuretic is needed.

Water-logged patients may be grouped as follows: (1) those in whom oedema is of circulatory origin or those with cardiac oedema, (2) those in whom oedema is of renal origin or those with renal oedema, (3) those in whom the oedema is in part of both origins or those with mixed cardiac and renal oedema, and (4) those in whom oedema is of hepatic origin or those with hepatic oedema. It is in group (1) that diuretics are most efficient, while in group (2)

they are least efficient and often inactive. In the latter, i. e., where oedema is of renal origin, there are such marked limitations to the effectiveness of diuretics that they are of almost no practical value. In group (3) diuretics are effective in varying degree dependent on the relative part played by the circulation in causing the oedema. In group (4) diuretics may produce diuresis, but as a rule no complete removal of the accumulated fluid is accomplished.

Often apparently entirely similar cases react very differently towards diuretics; quite often one diuretic may be effective and another ineffective in a given patient (see Chart I); while in a third patient though this holds true, the previously ineffective diuretic is effective while the formerly effective diuretic is almost inert. As yet no means of definitely prognosticating this last relationship has been discovered; it must be determined for individual patients by trial. Consequently, the failure to obtain a diuresis after the use of a given diuretic does not necessarily prove that the patient no longer is capable of diuresis; a different diuretic should be tried under these circumstances and this one may be effective.

Since diuretics increase renal work, and as there is much evidence to show that increased renal work on the part of an already damaged kidney leads to fatigue and a consequent depression of renal function, it has been considered desirable to give diuretics, except digitalis, intermittently rather than continuously. Digitalis, unlike other diuretics, appears to cause diuresis almost entirely, if not entirely, by improving renal circulation; consequently digitalis is given in the ways proved most effective on the circulation, i. e., usually over prolonged periods of time and not intermittently as is done with other diuretics. Digitalis, however, may be given in single or in several doses, repeated at short intervals, in total amount approximating the calculated effective dose, and this dosage may be subsequently repeated, thus digitalis being given intermittently rather than continuously.

It is in the water-logged cardiac that diuretics find their greatest utility. In my own experience this has been especially true of the patient beyond middle life with an hypertrophied heart, in which there are no organic valve lesions and whose cardiac rhythm is regular and rate but little accelerated. As such patients usually show a scant urine containing albumin and casts, often a decreased phthalein excretion and occasionally an increased blood urea nitrogen, they may be misdiagnosed as being patients with chronic nephritis and regarded as having oedema of renal origin. Diuretics may serve for such patients as a therapeutic test of renal efficiency and help in making a correct diagnosis, since if they respond with a marked diu-

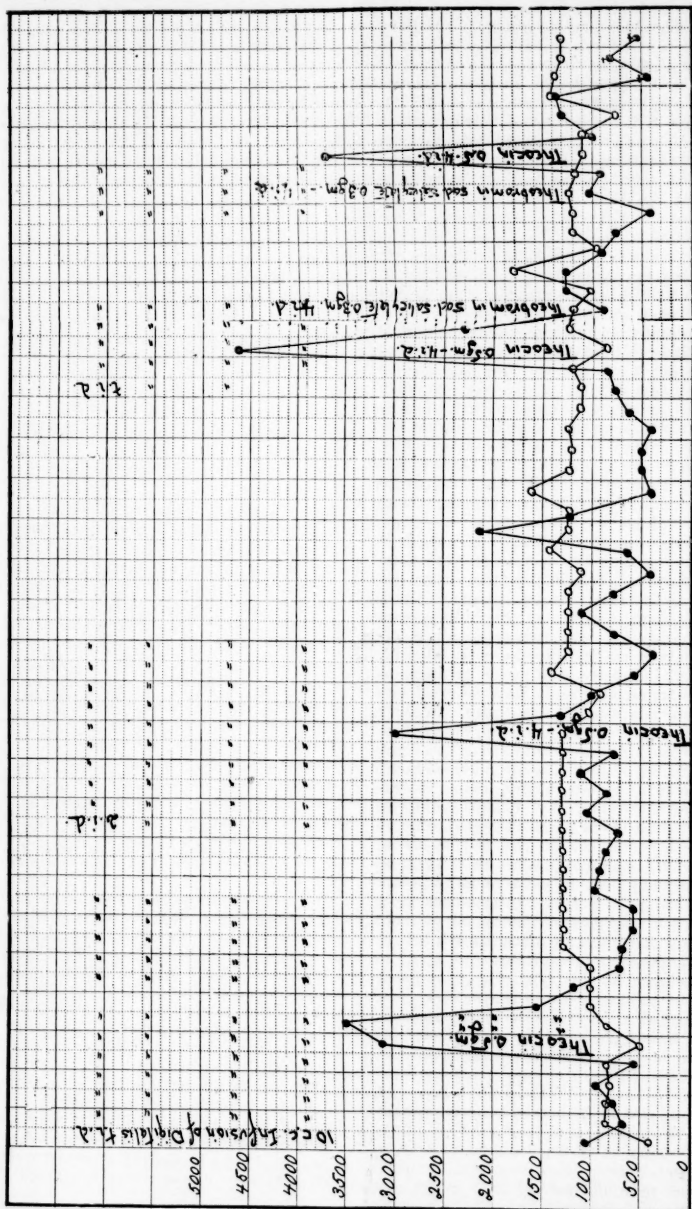


CHART I shows the diuresis produced by theocin in a patient with chronic myocardial failure and edema who failed to have a diuresis with theobromin sodiosalicylate. Subsequent to this the patient was given theobromin sodiosalicylate and a diuresis from theobromin sodiosalicylate, though theocin produced a prompt diuresis. In the chart the solid dots represent the urine output in 24 hour periods, and the circles the fluid intake.

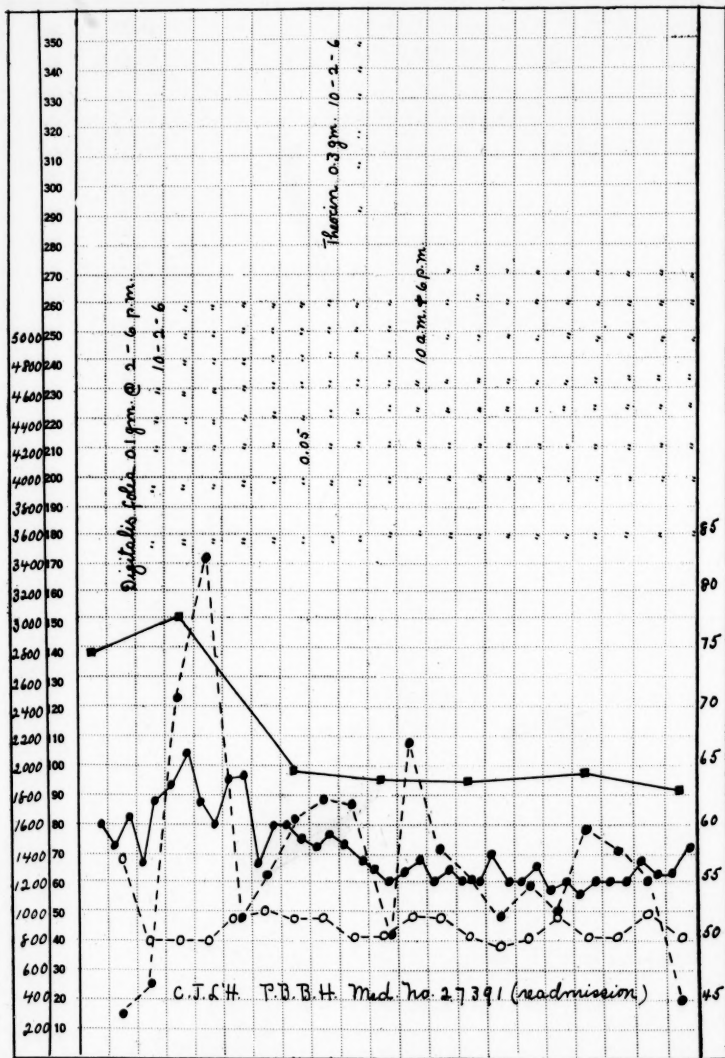


CHART III illustrates the diuresis produced in the same patient as shown in Chart II during a second admission. (For an explanation of the lines in the chart see Chart II.)

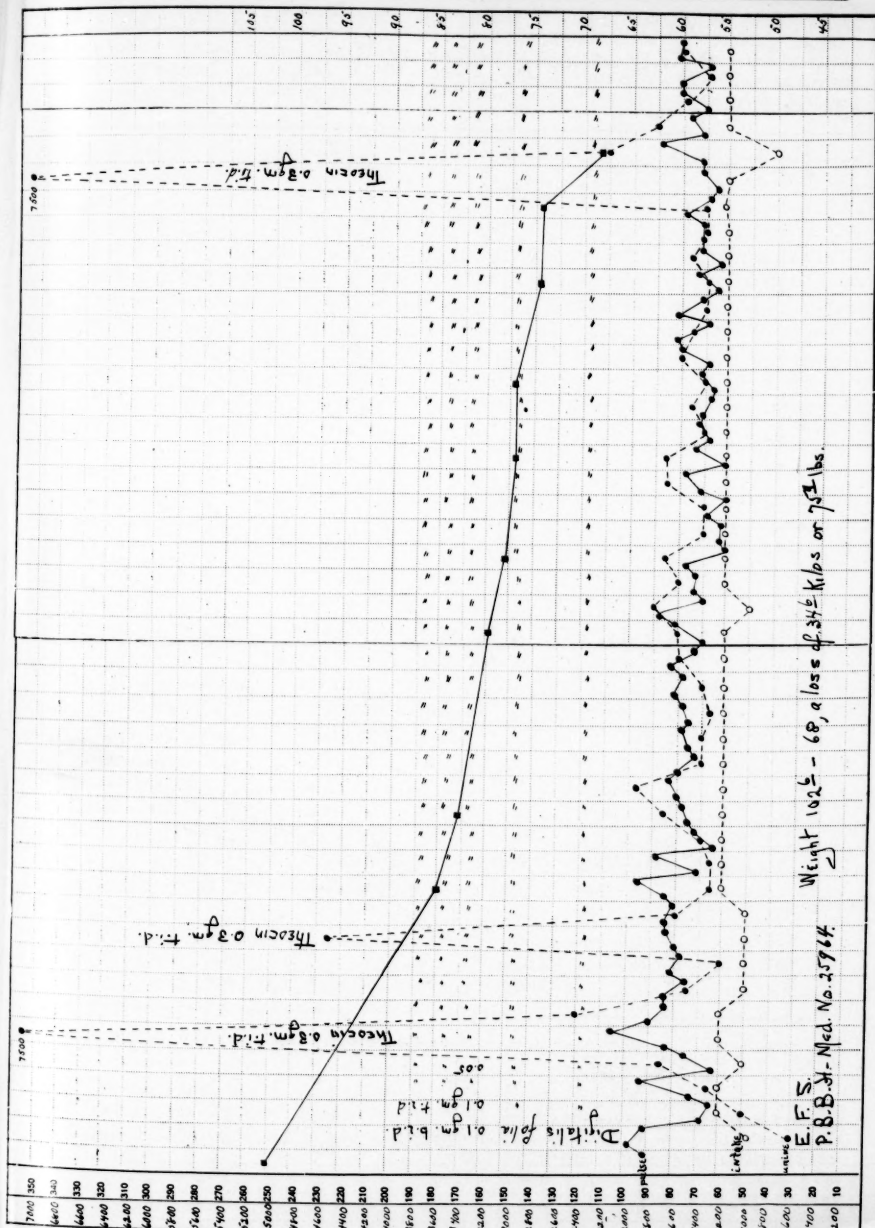


CHART IV shows the diuresis produced by theocin in an elderly patient with chronic myocardial disease and edema. (For an explanation of the lines in the chart see Chart II.)

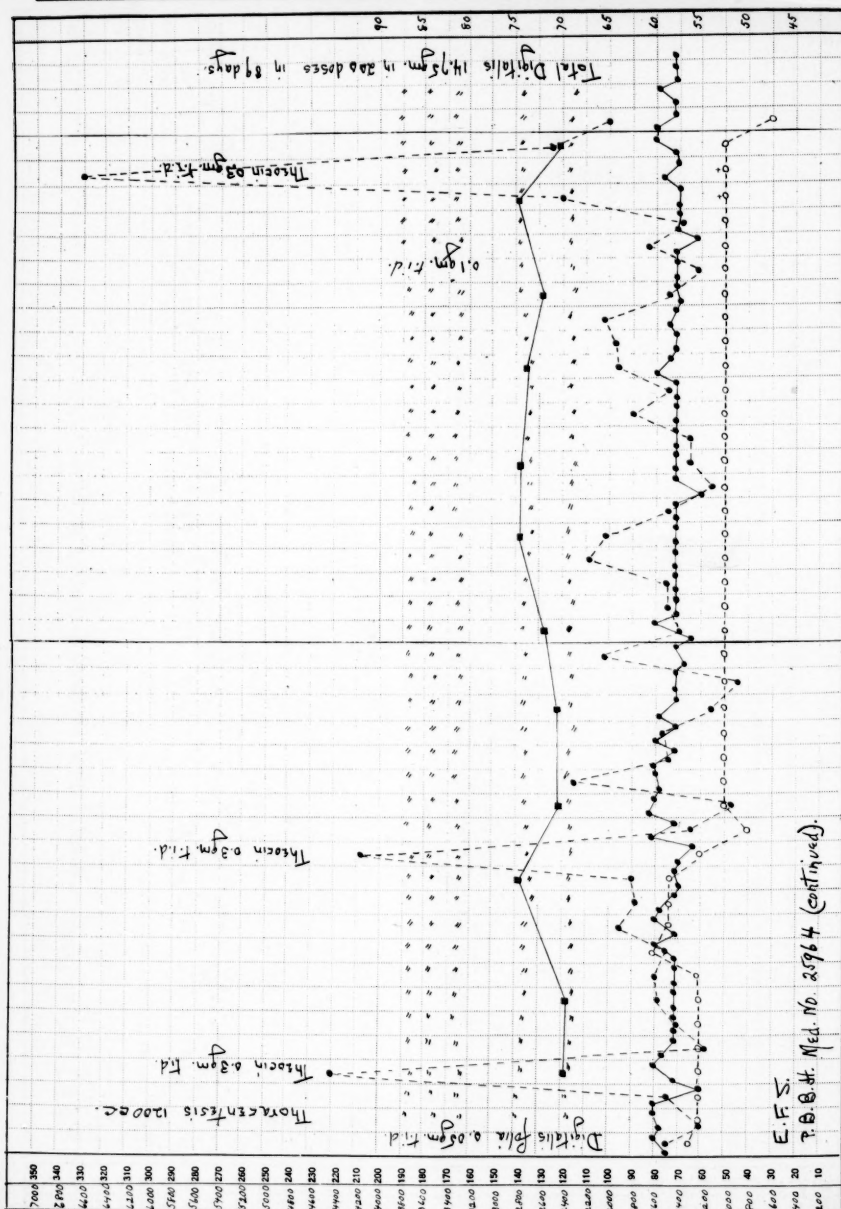


CHART V is a continuation of the same admission of the patient as shown in Chart IV. (For an explanation of the lines in the chart see Chart II.)

resis, the kidneys must be functionally efficient and it is not the inability of the kidney to excrete water that has caused the oedema, but rather the oedema has resulted in part from poor general circulation and in part from poor

pulse rate, fluid intake, urine output, weight and therapeusis as do the following charts, is from a man aged 65, who for seven years had had some dyspnea on exertion following a severe pneumonia. This did not incommode him very

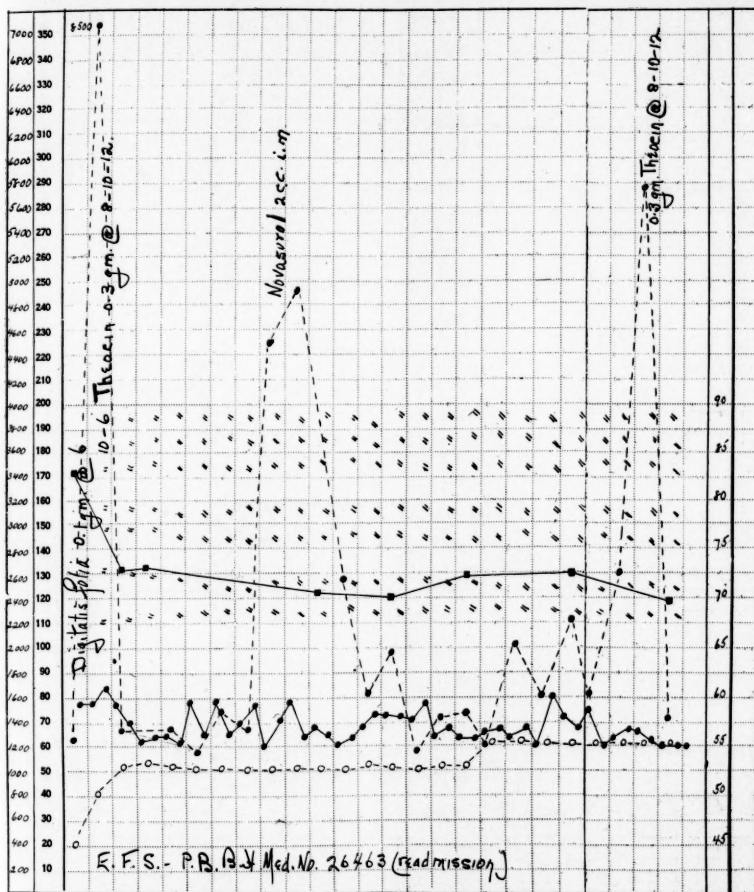


CHART VI shows the effects produced by theocin in the same patient as shown in Charts IV and V during a second admission. In addition novasurol, as shown in the chart, produced a striking diuresis at this time in this patient. (For an explanation of the lines in the chart see Chart II.)

renal circulation that has hindered renal function. Other types of cardiac disease also very frequently give excellent diuresis following the use of diuretics.

Let me illustrate some of these points by a few charts of cases studied by me at the Peter Bent Brigham Hospital. Chart II, which shows

seriously, however, until about five months ago, when his dyspnea became much worse and he developed oedema of the legs. On admission he had hypertension, a moderately enlarged heart with distant sounds, and no murmurs. His pulse rate was 105 on admission and 80 next morning; the rhythm was regular. He showed

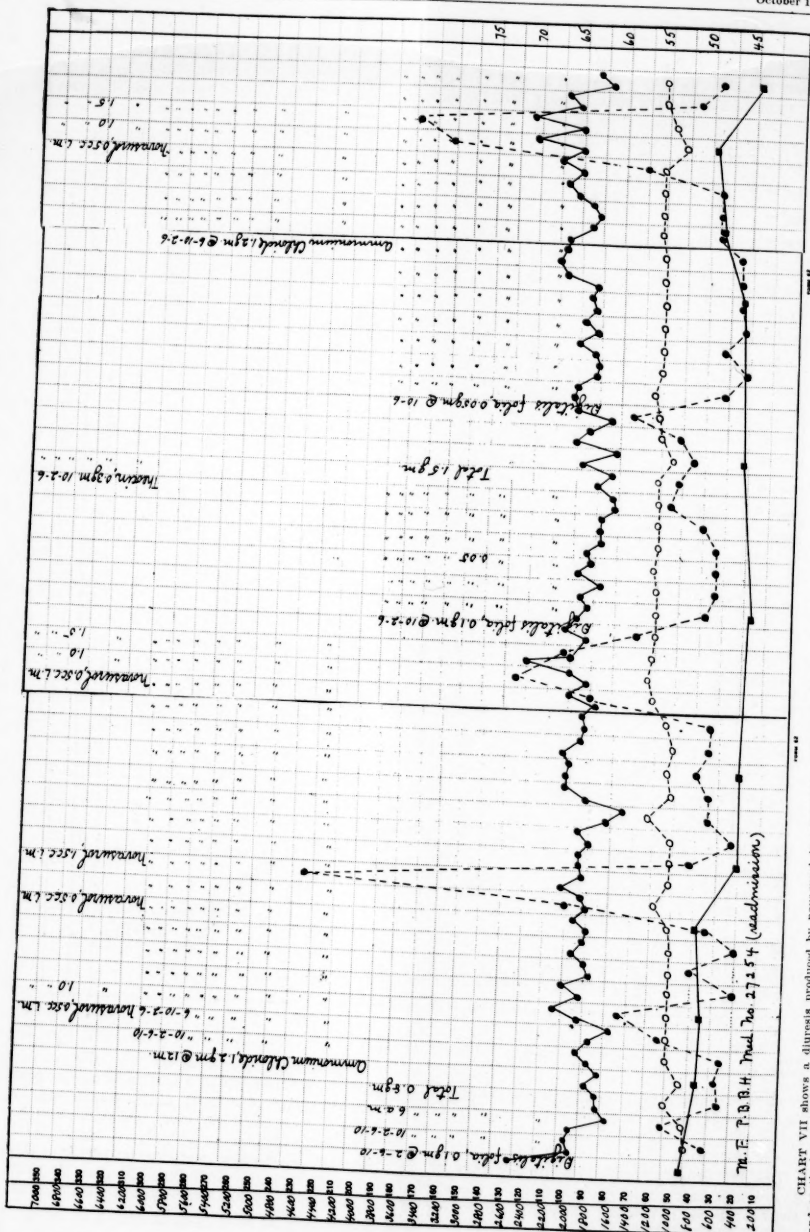


CHART VII shows a diuresis produced by tovastrin in association with ammonium chloride and digitalis in a boy of 17 with chronic valvular disease of the heart, mitral stenosis and aortic insufficiency. It will be noted that the diuresis is very little diuretic effect until after ammonium chloride had been given for several days. In this patient tovastrin produced no diuresis. (For an explanation of the lines in the chart see Chart VI.)

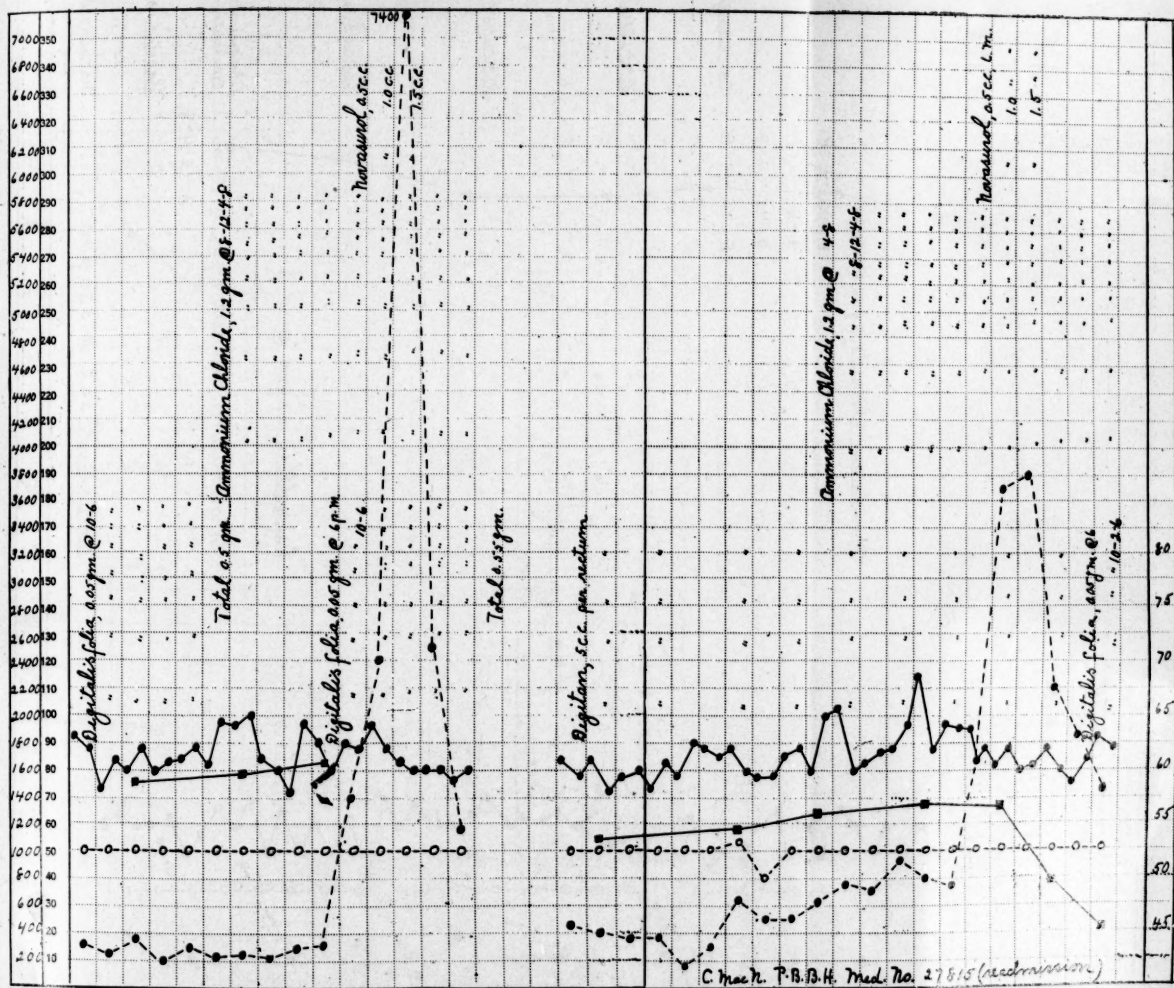


CHART VIII shows a diuresis produced by novasurol preceded by ammonium chloride and digitalis in an elderly patient with chronic myocardial disease and edema. In the chart two separate short periods during the stay of the patient in the wards are shown. (For an explanation of the lines in the chart see Chart II.)

marked generalized oedema and ascites. The urine was decreased in amount, contained a large trace of albumin and numerous brown granular and hyaline casts; the 'phthalein excretion was a trace in 2 hours and 10 minutes; the blood urea nitrogen was 32 mgm. per 100 c.c. of blood. This patient had been regarded as having chronic nephritis with oedema. His abdomen and thorax were tapped. Digitalis was given as shown in the chart. Forty-eight hours following completion of the first course of digitalis, theocin was given and produced a moderate diuresis. In ten days his weight dropped from 101.3 to 79.2 kilograms. Subsequently digitalis caused a diuresis, not increased by theocin. On a subsequent admission (Chart III) digitalis again caused a diuresis, not increased by theocin. At both times symptoms were very largely relieved and oedema disappeared. After treatment the urine showed only a very slight trace of albumin and a rare cast. 'Phthalein excretion increased from a trace to 20% in two hours and ten minutes, and blood urea nitrogen fell from 32 to 20 mgm. In one period of treatment this patient lost nearly 80 lbs. of oedema fluid. In this particular patient, with oedema very largely if not entirely of cardiac origin, digitalis alone was an extremely serviceable diuretic and theocin added little or nothing to the treatment.

Charts IV, V and VI show excellent diuresis from theocin and from novasurol combined with digitalis in another patient very similar to the previous one. He was a man of 58, who for two years had had dyspnea on exertion, which during the preceding two months greatly increased and was accompanied by swelling of the legs. On admission he was markedly oedematous, had high blood pressure and a much enlarged heart without murmurs. Cardiac rhythm was regular, rate 90. The urine contained a trace of albumin and numerous casts; 'phthalein excretion was a trace in two hours and ten minutes; blood urea nitrogen was 33 mgm. per 100 c.c. of blood. As shown by the charts (IV, V and VI) both theocin and novasurol caused very excellent diuretic effects. Novasurol was effective in this patient without antecedent ammonium chloride. The use of diuretics resulted in a prompt decrease in oedema.

In a third patient, one with chronic valvular disease of the heart, novasurol proved an efficient diuretic but only when preceded by ammonium chloride, as shown in the chart (Chart VII). In this patient theocin did not appear to produce a diuresis. He was a boy of 17, in whom symptoms had developed 2 years before admission. He had an enlarged heart with the murmurs typical of mitral stenosis and insufficiency and aortic insufficiency. His liver was enlarged, he had ascites and was oedematous. Treatment, as shown in Chart VII, removed his accumulated fluid and, though this was his fifth

admission, over a year later oedema has not recurred.

In a similar way, in another patient (Chart VIII) novasurol, along with small doses of digitalis was effective. The novasurol was preceded by ammonium chloride. This patient was a female, aged 60, with hypertension and chronic myocardial disease of the heart. Her heart was enlarged without murmurs; the rate was normal; the rhythm was regular, except for an occasional extrasystole. In her the treatment was very effective.

Chart IX is from a patient with alcoholic cirrhosis of the liver and ascites. In him novasurol preceded by ammonium chloride caused a diuresis, while theocin and euphyllin increased urine output but very little, or not at all. In other cases with ascites of hepatic origin I have had a similar experience; novasurol has been the most efficient diuretic, but it has not helped very greatly in removing the ascitic fluid of hepatic origin.

If we turn now to oedema of renal origin, diuretics of any sort are apt to be very much less effective as illustrated by Chart X of an oedematous woman with no evidence of cardiac lesion but having a marked picture of subacute or chronic nephritis. The chart shows therapy with digitalis, novasurol with and without preceding ammonium chloride, euphyllin, and a combination of high protein diet, thyroid extract and calcium chloride: none caused a diuresis. This patient also has received theocin without diuresis. In my experience it is decidedly rare for a diuretic to cause a diuresis when the oedema results from renal lesion, as in this patient. Not infrequently I have seen the urine flow depressed subsequent to giving a diuretic to a patient with renal oedema. I have never seen digitalis cause a diuresis except when there was evidence of cardiac pathology; consequently I have never observed digitalis to cause diuresis when oedema was due to renal disease or to hepatic disease.

The charts that I have shown are sufficient to illustrate the reaction of different types of patients to diuretics; they exemplify how a circulatory oedema decreases when a diuretic is given, how almost no effect follows their use when chronic nephritis causes the oedema and how with cirrhosis of the liver diuresis results but the fluid reaccumulates so rapidly that it is rare for a permanent decrease in the ascites to follow.

What may be said in regard to the several diuretics mentioned in the earlier part of this paper? Digitalis, as already emphasized, is a diuretic of a high order of efficiency in patients with cardiac disease; in some patients no other diuretic is needed, while in others digitalis alone does not cause an adequate diuresis. Digitalis is not a diuretic except in the presence of a cardiac insufficiency; when the heart is normal it

is not a drug that in any therapeutic sense acts as a diuretic.

Of the other drugs mentioned, caffeine, diuretin, theocin and euphyllin, are especially useful in conjunction with digitalis in patients with cardiac oedema. Of them caffeine is the least effective as a diuretic, and its use is limited by its effect on the nervous system, since beyond a certain amount it causes restlessness, excitement and sleeplessness. On the other hand, it definitely improves the pulse and may be regarded as one of our best circulatory stimulants. Furthermore, it is a stimulant of the respiratory center, and in large doses may convert a Cheyne-Stokes type of respiration into a regular rhythm. In these combined actions it has a definitely useful place in our therapeutics.

Theocin is a much more active diuretic than caffeine and as a rule more active than diuretin or euphyllin. I prefer it for cardiac oedema to all other diuretics, and have obtained best results in doses of 0.3 to 0.5 gram, repeated three times a day on one day and given again after an interval of twenty-four hours or longer, as judged by its effect. Unfortunately, in some people, it causes gastric upsets and these may be so marked as to preclude its use.

When theocin is not tolerated, I then use diuretin in doses of 0.5 to 1 gram three times a day or euphyllin in doses of 0.1 gram three to four times a day repeated after an interval as in the use of theocin. As already mentioned, in some patients one of these diuretics may prove effective and another ineffective, so it is always wise in case of failure after the use of one to try another. In general, diuretin and euphyllin are less efficient as diuretics than theocin. On the other hand, both have an effect on the coronary circulation, and in some patients with angina pectoris, bring definite relief in preventing or lessening anginal attacks. For this reason they may become the drugs of choice as a diuretic. Of the two I have had far less experience with euphyllin than with diuretin; further usage may demonstrate advantages for euphyllin not now recognized.

Any one of these four, caffeine, diuretin, theocin and euphyllin may be used in patients with oedema of renal origin. As a rule they produce no diuresis. However, if used in doses as described with subsequent periods without use, so as to prevent throwing undue work on the kidney, they are not harmful. If after several trials they fail to cause a diuresis, their use should not be continued. It is inadvisable to increase the dosage beyond that already indicated in case of failure to get a diuresis in renal cases. Rather it is preferable to try another one of the diuretic group.

Of all of our diuretic drugs novasurol seems to be the most effective. Its high content of mercury (33.9 per cent) leads to not infrequent development of signs of mercurial poisoning

(stomatitis, diarrhea, colitis, proctitis and renal irritation) following its use, and this necessitates caution in its use and often prevents continuation of it even when an excellent diuresis is caused. As some people are particularly sensitive to mercury, it is well to test this by a small preliminary dose of 0.5 c.c. given intramuscularly the day before giving the usual intramuscular therapeutic dose of 1.5 to 2 c.c. Again, very often its best therapeutic effects are only obtained after a preliminary course of ammonium chloride in doses of 1 to 1.5 grams three or four times a day, so that some delay is involved in its use when given in the manner likely to produce best effects. In my experience novasurol is the most effective of our diuretics for oedemas of non-cardiac origin, but its possible injurious effect on the kidney demands very great caution in using it in patients with nephritis. Perhaps it is wiser not to try it when the oedema is clearly caused by nephritis. In cirrhosis of the liver it may be used in the manner indicated above without undue risk of injury to the patient. Occasionally it is a very useful addition to the therapy of cirrhosis of the liver. Possibly in some of these patients its effect is enhanced by the mercury moiety of the drug in the role of an anti-syphilitic drug.

It has seemed worth while to present this discussion of diuretics because experience seems to show that many of our membership are not utilizing the diuretics as much as their effectiveness in properly selected patients justifies, for they are effective and useful drugs. Particularly in some of the water-logged cardines are the results of their use very striking. In my own cardiac patients with the use of digitalis, supplemented by diuretic drugs when the oedema persists, I find that there is no need to use cathartics as a method of eliminating fluid accumulated in the form of oedema, and it has seemed to me that the patients are less debilitated than when they have frequent watery bowel movements as the result of vigorous catharsis. In my experience, digitalis plus diuretics is fully as efficient in the treatment of cardiac disease with oedema as digitalis plus watery catharsis, and it is much more comfortable and less debilitating to the patients. I would make a plea for the abandonment of the time-honored custom of giving our cardiac patients large doses of magnesium sulphate, compound jalap powder or calomel in addition to digitalis, and substitute for the cathartics some of these diuretic drugs in case the oedema does not rapidly diminish with digitalis alone.

CHAIRMAN SMITH: It seems to me we have had four rather unusual papers on the kidney. I am going to ask Dr. A. A. Hornor to open the discussion.

DR. W. B. BREED (Boston): Dr. Hornor is not here; he asked me to open the discussion.

We ought to be very thankful to Dr. Ohler

for the interesting and important matter that he gave us, and particularly for bringing our attention back to the importance of urinalysis historically. Urinalysis was the first physical and chemical test that we had in medicine, coming as it did directly after the ordinary clinical examination.

The twenty-four hour examination of urine has been very much overdone. Since diabetes has been studied so closely, the twenty-four hour specimen has been overemphasized. In the first place you do not get the very important information as to when you have glycosuria. Also in relation to nephritis you are very apt to miss showers of blood cells, and the sediment after twenty-four hours is obviously never fresh. More frequent examinations of single specimens, especially those in relation to meals are to be desired.

There is one thing that Dr. Ohler did not mention and that is the fact that various substances in the urine give a false positive with Benedict's solution. The qualitative solution is adequate and necessary in the treatment of diabetes, but in ordinary urinalysis of single specimens in routine cases there are more than a few false positives. The principal substances causing this false reaction are albumin and various salts. The concentrated urines also often give false positives, and there is consequently a good deal of confusion as to whether or not patients have a true glycosuria.

Another point about the teaching of urinalysis to diabetic patients;—it seems to me that it is not really essential to teach them the examination for diacetic acid, because except in very rare cases if a patient is sugar free he is not suffering from acidosis, and diabetic patients have troubles enough in keeping themselves sugar free with their chemical tests. One may prevent them from becoming neurotic by not giving them a test for diacetic acid.

We all have heard of the aseptic conscience that the surgeons speak of, and I should like to make a plea that we follow Dr. Ohler and try to develop a urinalysis conscience.

(Applause.)

DR. J. H. PRATT (Boston): I am pleased that Dr. Ohler has called attention to the value of the dilution and concentration test, as I have used it for a number of years in the study of renal function and regard it highly. It makes greater demands upon the kidney than does the ordinary two-hour test of Schlayer or Mosenthal, and hence one usually obtains urine of lower specific gravity and urine of higher specific gravity in the dilution and concentration test than in the two-hour test. This makes interpretation of the results easier. For the past two years I have used a slight modification of the method. It consists in giving no food on the day of the test

until 11 A. M.; otherwise, I employ the test as described by Dr. Ohler on the printed form he has passed about.

Inability of the kidney to secrete a concentrated urine is the first sign of renal insufficiency in chronic Bright's disease. Hence the importance of detecting this loss of concentrating power on the part of the kidney. I use a simple concentration test when for any reason the method already described is not used. The patient is instructed to drink no fluid after 5 P. M. and to eat no breakfast the following morning. Urine is voided at 9 A. M. and 11 A. M. Normally the specific gravity should reach 1025 in these concentrated specimens. If the maximum specific gravity is 1020 or less, it indicates a lessening of the concentrating function. An increase in the non-protein nitrogen of the blood occurs only late in chronic kidney insufficiency. Usually long before there is any significant increase in blood urea in chronic nephritis, there is a failure on the part of the kidney to secrete urine of normal specific gravity. Inability of the kidneys to secrete nitrogen in normal amount bears a causal relation to the azotemic type of uremia. Normally the percentage of nitrogen in concentrated urines rises to one per cent. or more. If the concentrating function of the kidneys is impaired, the percentage of nitrogen in the most concentrated specimen of urine obtainable may be much less than one per cent. I have been studying the maximum concentration of nitrogen in nephritis. I cannot yet speak of its value as a diagnostic test. I believe, however, that the simple specific gravity test is of more value than the time-consuming nitrogen determination. I am confident, however, that the nitrogen concentration test will be of help to the surgeon in the diagnosis of unilateral kidney disease. It is possible to make this test with as little as 1 c.c. of urine. In one case in which determination of the nitrogen concentration in the urine was obtained with catheters in the ureters I found a normal concentration of nitrogen in the urine from the left kidney and only one-tenth as great a concentration from the other kidney, which was excreting practically no nitrogen. The result was of real diagnostic aid, as blood in the urine prevented the surgeon from getting any help from the phthalein test.

Dr. Smith states that he believes ptosis and intestinal indigestion produced pyelitis or were factors in the etiology. He cited, in support of his view, a case in which, after some intestinal disturbance, the pyelitis became worse. The intestinal symptoms may have been a toxic manifestation of the pyelitis. I do not think the conclusion justifiable that the upset in intestinal function necessarily leads to reinfection of the pelvis of the kidney.

I wish to endorse what Dr. Christian said in regard to the superiority of inducing diuresis rather than catharsis in the treatment of oedema.

DR. J. D. BARNEY (Boston): Mr. Chairman, I think the urologist has a great advantage over men in other lines of work in that he has so many methods of making an accurate diagnosis, and as evidence of that we took account of stock one year accurately in the Urological Clinics at the Massachusetts General Hospital and found that our error was only 4 per cent. in the pre-operative diagnosis, which seemed to us pretty good. Those, of course, were operative cases. Where there was no operation performed we had no means of telling whether we were right or wrong except by continued clinical observation of the case.

I think it is important for the medical man and the surgeon as well as the urologist to realize that a kidney lesion may simulate disease of other organs in a great many cases. Kidney lesions which may simulate appendicitis, or gall-bladder disease, or gastric disease, or duodenal disease, or pancreatitis, may exist and may be rather difficult to make a diagnosis on, the reason being that the urine in these cases is sometimes entirely negative and persistently negative, and because an x-ray may not be done at once or may not show the actual conditions present.

An x-ray may show a stone but it must be remembered that sometimes stones do not show. I have seen several of them which did not show at all and others which did not show for a long time. I acknowledge they are rare, but they do exist.

Then, also, the simple x-ray is not sufficient because, as Dr. Smith pointed out with his lantern slides there are conditions which exist in the kidney pelvis which only a pyelogram or ureterogram will disclose, so that in any obscure case where we think the kidney is at all suspicious, these examinations may well be undertaken.

Another condition which I think should be laid stress on is that of aberrant renal vessels. These do not show in the x-ray. The pyelogram may or may not reveal them. We often see cases where there is a distinct break in the continuity of the pelvis and the ureter and where we think very definitely that there is an aberrant vessel present. We see cases where there is no break in the continuity and we do not suspect it but it is very common to find these aberrant vessels. If they go on for any length of time, they are the cause of infection, with ultimate destruction of the kidney.

I won't go into the treatment of those conditions because they vary in each case, but they are very common things and it should be remembered that they are very frequently bilateral. I have often seen the kidney on both sides dilated and destroyed to a certain degree by these aberrant vessels so that in operating upon such a kidney, conservatism should be employed if possible, the idea being that the other side is similarly affected and one may have to operate ultimately upon the second side.

Another thing I think would be worth stressing is that the various tests for tubercle bacilli in the urine, although they are very accurate, still may be in error. That is particularly true of the microscopic test of the sediment and it is also true but to a lesser extent of the guinea pig test. I have seen several cases which ultimately proved to be renal tuberculosis where on various occasions the sediment showed no tubercle bacilli even after careful search and where one or two and sometimes three guinea pig inoculations, all done properly, were negative.

Sometimes one can explain these discrepancies and sometimes one cannot, so that I think in case of any dysuria, pyuria, and hematuria, one should always keep in mind the possibility of renal tuberculosis.

Repeated study of many cases is necessary in order to arrive at a correct conclusion. That is true, of course, of the suspected cases of renal tuberculosis and it is true in certain other conditions. For instance, the pyelogram on the first occasion gives one picture. It may not be satisfactory or conclusive, and another series of studies, as Dr. Smith pointed out, may show an entirely different picture, due perhaps to the fact that on the first occasion the pelvis was not filled completely or properly. I find many times that we are rather expected to give a definite diagnosis of considerable refinement on the first occasion but it is not always possible so that a study on several occasions sometimes is necessary in the difficult cases.

Dr. Bugbee brought out well the immense importance of the blood chemistry and phenolsulphonphthalein test. We certainly could not get along very well without these tests, but at the same time, I think we should all bear in mind the fact that one's clinical judgment should be exercised as well. I have seen cases where the blood chemistry was perfectly satisfactory and where the phenolsulphonphthalein was satisfactory, but where I did not think it was safe for the patient to be operated upon. The tongue was dry or coated, the appetite was poor, and the patient did not feel quite up to par, so I think that in spite of what the laboratory says, we should stop and consider a little what our clinical judgment, which after all is the fruit of experience, tells us and remember that in all these cases, especially the prostatic, it is better to drain them a few days too long than a few days too little. (Applause.)

DR. CHANNING FROTHINGHAM (Boston): From the result of some experimental work done a few years ago by Dr. Christian it was suggested that diuretics in acute nephritis may be actually harmful rather than of benefit. Furthermore, it seems common sense to try to put at rest an acutely inflamed organ rather than to try to stimulate it to action. At the present time in the literature on renal disease the usual advice

is against the use of diuretics in acute nephritis and also in many cases of chronic nephritis. I can't help being impressed, however, as I see cases with physicians in various communities with the fact that these practitioners feel that frequently they have improved the output of urine and also the patient's condition during acute or chronic nephritis by the use of different types of diuretics. I should like to ask the speakers if they have any definite data which will help us to decide whether we ought to use diuretics in acute and chronic nephritis or not; and also whether, even if they do no good, they do any actual harm.

DR. NILES: I had a case of colitis. The roentgenograms were taken on that basis and showed nothing in the kidney at all. With further study Dr. George took another x-ray and found a small stone about 2 c.c. below the left kidney and on that same side there was a general colitis of the large colon, the colon being very much constricted so that there were symptoms of colitis although that wasn't the real trouble. I wondered whether it is common to find a colitis in conjunction with the stone in the ureter.

DR. R. F. O'NEIL (Boston): I regret that I did not hear the first two papers. It is always a pleasure to hear a paper by Dr. Bugbee and I think we are to be congratulated on his bringing up again this very important subject of renal function and blood chemistry. These procedures have long been appreciated by the genito-urinary surgeons and have been of the very greatest use to us in our prognosis and as to when we shall or shall not operate. I should like to call attention to the fact that I think it is quite as important for the general surgeon to employ these methods as it is for us. There are many cases which come to operation which have some concomitant lesion of the kidney which, although not perhaps the main feature in the case, is of sufficient severity to seriously interfere with the end recovery or to bring about a stormy convalescence.

I am quite sure that some of the fatalities which are put down to ether or some other cause are really due to renal insufficiency and the old methods of dessication of patients before operation have given way to forcing fluids up to the last minute. The general surgeons largely through the efforts of the genito-urinary surgeons more and more appreciate the importance of the study of the kidney in any surgical procedure. It is now routine at the General to have an N. P. N. estimation and renal function done on every case.

Where these vary from the normal, a few days of treatment by diuresis and Digitalis, and the postoperative course is very much smoother.

There is one feature that Dr. Bugbee brought out I should like to emphasize particularly which is of comparatively recent development

in the pthalein estimation and that is the fifteen-minute period estimation. You get your total function at the end of one hour and two hours and you say "Fine, 50 or 60 per cent.!" That case is operated on and does not do well. I believe that if in such a case the pthalein had been estimated at fifteen-minute periods you would have found that although the total function at the end of two hours was good, you got your maximum output at the end of three-quarters of an hour or an hour, showing a damaged kidney which was not suspected.

I think this goes far to explain some of the cases that Dr. Barney spoke of where from a laboratory point of view the patient is all right but from the clinical standpoint he is not.

I have heard of a case within a day or two where a simple pyelotomy was done on a man who was said to have good N. P. N. and good renal function. This patient went on to complete anuria for a number of days. I believe if the pthalein had been estimated at shorter periods, it would have shown a delayed renal function and therefore a damaged kidney where perhaps with longer pre-operative treatment the convalescence would have been much easier.

DR. WILLIAM R. OHLER (Boston): It has been very interesting to me to find out how much really can be learned simply by the urine examination. The difficulty lies in getting the examination done. If examinations can not be done in a physician's office, we have at our disposal several hospital laboratories and several good commercial laboratories. In addition it is surprising how many drug stores now advertise that they do urinalyses.

It strikes me that this sort of drug store advertising is in the nature of an indictment against the medical profession, and that it is time for the profession to assume the responsibility for such a fundamental study and not allow this to be done in the corner drug store.

DR. GEORGE GILBERT SMITH (Boston): Dr. Pratt has asked me to prove a thing which is rather difficult of actual demonstration. If we were like the Fisheries Commission and could put a tag on the end of a colon bacillus in the bowel and catch him coming out through the urinary tract, we could prove it, but, unfortunately, we cannot do that. However, I think argument by analogy and process of reasoning supports very well the belief which is generally held by urologists, that the intestinal tract is the most common portal of entry for colon bacilli into the urinary tract.

It has been shown that the lymphatics from the ascending colon communicate directly with the pelvis of the right kidney. It is probable also that through breaks in the mucous membranes of the intestine, which accompany a mild ulcerative process, the colon bacillus gets into

the circulation and is then passed out through the kidney.

It has been found in the study of cases after operation that at least a third of these cases have colon bacilli in the urine but if the drainage of the urinary tract is normal, the mucous membrane is not infected and the organisms simply pass through.

With ptosis, it is well known that the kidney is rendered more vulnerable. In the first place, the drag of the ptotic kidney on the renal vein tends to set up a congestion in the kidney and a certain amount of venous stasis. In the second place, often—though not always, of course—there is some obstruction to drainage of the pelvis, as was demonstrated in one or two of the plates that I showed. There may be another factor besides ptosis; I think there has to be. In other words, the ureter must have a fixed point so that when the kidney falls a kink is formed at the point where the ureter is fixed or else, as Dr. Barney has said, there must be an aberrant vessel, but it is a fact that in many of these cases there is an obstruction to drainage which accompanies ptosis.

A normal kidney, if infected, will clear itself of the infection if the patient is simply kept quiet and given plenty of water, but if there is an obstruction to drainage, conditions exist which prevent clearing up of infection the same as they exist in any closed cavity which is infected and cannot drain.

We have seen many of these cases where the history of the urinary infection has been preceded by a digestive upset. By that I mean a period of constipation followed by a diarrhea, perhaps, and the symptoms of a mild colitis. Such cases often come on when the patient has been out of his routine of living, been perhaps on a vacation, traveling a lot, eating a lot of unaccustomed food, and probably not taking great care of the clearing out of the bowel.

In regard to Dr. Niles' question, I have seen one case recently where an infection of the lower pole of the kidney by a septic infarct was followed by perinephritis which involved the peritoneum and the peritoneal covering of the colon where it was intimately adherent to the kidney, but I think such cases are very rare and I would say that in general a renal stone or ureteral stone is not accompanied by symptoms of colitis.

DR. HENRY G. BUGBEE: I wish to express my appreciation at hearing the papers of Doctor Ohler, Dr. Smith, and Dr. Christian. I have enjoyed them and I have enjoyed the discussions.

DR. HENRY A. CHRISTIAN (Boston): In speaking to you I think I forgot to say that, in connection with these patients, as illustrated by the first case in which renal function shows evidence of kidney disease as shown by the presence of albumin and the lowered 'phthalein and

increased blood urea nitrogen, when you improve the circulation and remove the edema, there is always a striking improvement in all of these factors indicating poor renal function, and if your studies are made after treatment, you are not likely to make the diagnosis of nephritis, which has seemed justified by your studies before treatment.

In regard to cathartics, I wish to emphasize that I was referring to the use of cathartics in place for the use of cathartics in edemas of other patients with cardiac edema. There is a definite types, notably in cirrhosis of the liver with ascites and also in some of these cases with nephritis and edema. I would not condemn cathartics, but limit their use to those cases in which diuretics do not effectually replace them in eliminating fluid.

In regard to acute nephritis, my own feeling is that the diuretic drugs are best omitted. Simply the use of a moderate increase in fluid intake is the safest diuretic for acute nephritis. We should remember that, with few exceptions, patients with acute nephritis get well, and as they get well, their urine output always increases, whether you give them this, that, or the other form of therapy; consequently it is very difficult to judge of therapeutic effects in them. Remembering that they practically always get well, I use no diuretics whatsoever.

Finally, in closing, let me bring to your attention that in the lovely months of May and June in England, just a hundred years ago, Richard Bright was completing the correction of the proof and putting through the press his monumental study, which finally attached to the presence of albumin in the urine and edema a definite significance, and from that time on, i. e., since 1827, we have recognized nephritis as an entity that could be diagnosed by the clinician from a study of the patient and the urine.

In August, 1827, there appeared from the printers his publication, which included not alone a study of renal edema, but also of hepatic edema, cardiac edema, ascites due to portal obstruction and other forms of edema, a study that associated these lesions with the clinical entities that here a hundred years later we are still studying. (Applause.)

CHAIRMAN SMITH: That completes our program and I declare the meeting adjourned.

The meeting adjourned at twelve-thirty o'clock.

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN
WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.

F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 13411

STOMACH TROUBLE AT FIFTY

MEDICAL DEPARTMENT

An American broker fifty years old entered February 12 complaining of epigastric pain and vomiting.

Eleven months before admission he began to have a great deal of gas three hours after meals. With the first attack he was nauseated and vomited a pint of brownish fluid. He then had a sour taste in the mouth. He took milk of magnesia with complete relief. He stayed in bed six days. Attacks of gas kept recurring three times a day soon after meals but without nausea or vomiting. He continued however to have mild pain in the midepigastrium, always relieved by milk of magnesia and by food. A month after the onset he began to have sharper pains, sometimes beginning in the chest or the small of his back. With these attacks he vomited brownish liquid, no blood. He slept very poorly. Occasionally the pain woke him. Six months before admission he was able to keep down nothing but white of egg. He gradually increased his diet without any vomiting, and for three months had no more trouble except an occasional midepigastrie twinge relieved by milk of magnesia. Then the pains returned, more persistent and not relieved by milk of magnesia. After a week he again had nausea and vomiting. November 2 X-ray examination in the Consultation Clinic showed the stomach in median position, normal in outline and freely movable, with vigorous peristalsis. The region of the sphincter was irregular. The duodenal cap showed constant irregularity in outline and slight delay in the first portion. At the six-hour examination the stomach was empty. The head of the meal was in the hepatic flexure. The cecum was normal. After two months more of nausea and vomiting the patient went to a physician who made a diagnosis and put him on a diet. He was not relieved. Three weeks before admission he returned to the physician, who put him on a stricter diet, again without relief. Throughout the illness his appetite had been very poor. His stools were fluid and occasionally very dark. Although he had felt weak and had lost 60 or 70 pounds, he continued to work

at his business, which taxed his nerves and worried him.

His father and mother both died at seventy-five of shock. One sister died possibly of purpura.

At twenty he had Neisserian infection and pyuria. Twenty years before admission he had sore throat and tonsillectomy. His bowels were always somewhat constipated.

Clinical examination showed a poorly nourished, poorly developed man vomiting and complaining of epigastric pain. Tongue dry. Slight pyorrhea. Barrel chest. Lungs hyperresonant. Exaggerated expiration. Location of the apex impulse of the heart not recorded. Left border of dullness 11 centimeters from midsternum, 3 centimeters outside the midclavicular line. No other enlargement to percussion. Sounds and action normal. A systolic murmur at the apex and base. Pulses normal. Blood pressure 180/100 to 85/53. Abdomen scaphoid. Slight midepigastrie tenderness. Liver edge 3 centimeters below the costal margin in the right mammary line, smooth, not tender. Upper border of liver dullness at the fifth interspace. External rosette of hemorrhoids. Right epididymitis, left questionable. Coarse tremor of the hands. Slight Heberden's nodes. Pupils and reflexes normal.

Before operation urine normal in amount, very cloudy and alkaline at one of two examinations, specific gravity 1.020 to 1.026, ferric chloride and sodium nitroprussic acid positive at one, 1 to 5 leucocytes per high power field at both. Renal function 55 per cent. Blood: 23,700 to 11,000 leucocytes, 83 per cent. polynuclears, hemoglobin 70 per cent., reds and platelets normal. Wassermann negative. Stool and two specimens of vomitus showed no gross blood, guaiac negative. Gastric analysis: fasting contents 28 cubic centimeters of acrid dark brown watery material, guaiac positive, free acidity 38, total acidity 55; test meal 65 cubic centimeters of acrid white watery material loaded with coarse sediment, guaiac negative, free acidity 64, total acidity 88.

X-ray. The stomach appeared normal in position. Peristalsis was active and deep. There was no retention at the end of six hours. There was a persistent irregular filling defect involving the prepyloric portion of the stomach, the pyloric sphincter and the base of the first part of the duodenum. There was considerable tenderness directly over this area. At six hours the motor meal had reached the cecum. The deformity had increased somewhat since the Consultation Clinic observation in November. Plates of the kidney region and gall-bladder showed no evidence of pathology.

Before operation temperature 99° to 100°, pulse 83 to 101, respirations 15 to 28.

The patient continued to have considerable epigastric pain and some pain between the shoulder blades. This quieted down under atro-

pine. He continued to have nausea from time to time. February 14 he vomited 40 ounces of milk and cream mixture. He was given a subpectoral and Murphy drip. After this he took the Sippy mixture fairly well until midnight February 15, when he again began to vomit and to have dull aching high in the abdomen, which continued to be quite soft. Soon after returning from the X-ray examination he complained of severe generalized upper abdominal pains such as he had never had before. There was definite rigidity of the right rectus. The left remained fairly soft. The entire abdomen was fairly rigid. Two surgeons and a physician in consultation advised immediate operation. The mortality risk was estimated at 25 per cent.

February 16 operation was done. Two days later the patient was in fair condition and not vomiting. The pulse was irregular and rapid. There was a pulse deficit of 70. He was given intramuscular digifolin and quinidine. By the 20th the discharge from the wound had increased and the surrounding skin was red. The pulse was regular. Preparations were made for a transfusion the night of February 21. There was continuous oozing of blood through the wound. The blood pressure fell to 85/53. Spasm and tenderness of the entire right side developed. The leucocyte count was 27,400. He was given subpectorals, caffeine and digifolin. The evening of February 22 he died.

DISCUSSION

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY

The history of three months' remission of symptoms is very important if true, so I give notice that I am taking it as true, hoping that the record is correct.

The irregularity of the sphincter does not show very well on the X-ray plate. I take it that at this time they are thinking of duodenal ulcer. Most of his symptoms have pointed in that direction. Especially after three months' remission of symptoms the chief lesions that you and I are thinking of are ulcer and cancer. Ulcer almost always has remissions: cancer very rarely does. Then most of this time the pain came at irregular intervals and seemed to be relieved by food or alkali. But there was a period when it was not so relieved. On the whole, up to the time of this first X-ray it is our belief that he probably had duodenal ulcer.

Of course the poor appetite is against the diagnosis of duodenal ulcer. Usually there is good appetite. This point is in favor of the diagnosis of cancer, and we have to keep those two steadily in mind to the very end of the case. We see loss of weight with either disease, especially when we have a reduced diet, but it is commoner with cancer than with ulcer.

The past history does not seem to have any bearing on the present. We are dealing wholly with a stomach illness, so far as I see, up to this point. I should suppose he had a hypertension before he came in, with hypertrophy and dilatation of the heart, and nothing else. But I do not believe it has anything to do with his stomach symptoms. Also we get symptoms a little like these with uremia, but so far we have no good reason to consider that.

NOTES ON THE PHYSICAL EXAMINATION

There is no evidence of cancer in the liver, no evidence of passive congestion. It may be a normal liver.

The coarse tremor of the hands makes you wonder about alcoholism. Nothing has been said about it so far. That goes with some types of stomach trouble, but not with anything recorded here, not with pain that comes at irregular intervals and is relieved by food and soda. Alcoholic gastritis is a rather rare disease. It does not present these symptoms.

The positive ferric chloride and sodium nitroprussic acid probably indicate some organic acids in the urine from starvation and vomiting.

The gastric analysis favors ulcer rather than cancer.

He did not take forty ounces of milk and cream mixture all at one time, so the vomiting February 14 shows some retention at that period.

"Soon after returning from the X-ray examination he complained of severe generalized upper abdominal pains such as he had never had before." Of course we are thinking of perforation, either of ulcer or cancer.

DIFFERENTIAL DIAGNOSIS

I am still in doubt between cancer and ulcer, the two diseases, and the only two so far as I know, that we ought to consider in this case. Let us go back and sum up the evidence. In the first place if it is true that he had no symptoms until forty-nine, that is older than most people are when they begin to have symptoms from ulcer. So the age, taken in connection with the short history, not years but months, favors cancer. The relief from magnesia favors ulcer, as does the high acidity. I should say the pains that seemed to be in his chest and back are hard to explain either way, but would go a little better with cancer than with ulcer. The complete relief for three months favors ulcer. The fact that the earliest lesions were in the duodenum strongly favors ulcer. Cancer of the duodenum is a rare lesion. It might perfectly well have been an ulcer that extended from there up through the pylorus. It could not easily have been a cancer that extended from there back into the stomach. His appetite has been poor; that favors cancer. The fact that he has been able to go on with his business

is commoner certainly with ulcer than it is with cancer.

In the physical examination there is nothing of any importance that I see except the acidity in the stomach and the X-ray. They felt no lump. They got no anemia. They got nothing that is characteristic in any other way that I see. Now in the end we have perforation. Which is more apt to be perforated, ulcer or cancer?

STUDENTS: Ulcer.

DR. CABOT: Cancer perforates too, but ulcer perforates often.

Putting the evidence all together, no one can say it is a clear case either way. On the whole I vote for ulcer. I vote chiefly on the ground of the remission, of the position of the earliest X-ray lesion and of the perforation, together with the high acidity. But I see difficulties with that. It would not surprise me at all if I were wrong. However, I believe that is the diagnosis under which they operated.

PRE-OPERATIVE DIAGNOSIS

Perforated duodenal ulcer.

OPERATION

Gas-ether. Free barium was encountered in the peritoneal cavity, but not a great deal of fluid. The perforation was located in the second part of the duodenum near the posterior wall. The posterior wall of the ulcer was apparently formed by the head of the pancreas. The duodenum was infolded in two layers over the perforation and the omentum was stitched over this. A cigarette wick was inserted.

FURTHER DISCUSSION

His heart is doing badly. We knew it was enlarged before, presumably on the basis of a hypertension. He probably has a hypertensive type of heart trouble.

What we are afraid of now is digestion of the wound by pancreatic juice from a pancreatic fistula. But we have no positive proof of it yet.

He died of peritonitis, I think. I think Dr. Mallory will say it was a perforated duodenal ulcer with more recent trouble, localized peritonitis. There will be a cardiac hypertrophy and dilatation with some arteriosclerosis, but without any important changes in the kidney or anywhere else. I do not believe there will be anything in the lungs or the nervous system.

A STUDENT: How frequently does cancer perforate?

DR. CABOT: I should say off-hand once in a hundred times. I remember four or five in my necropsy experience here.

A STUDENT: How did you differentiate duodenal from gastric ulcer?

DR. CABOT: There are no symptoms of duodenal separate from symptoms of gastric. They

used to say so. I do not believe it can be done. It is wholly a diagnosis of X-ray. We have not heard the last word yet. The trouble may be in the stomach.

A STUDENT: Do you think cancer of the head of the pancreas could give these symptoms?

DR. CABOT: I do not see how. That disease remains latent, without giving symptoms for a while. Then it gives two symptoms, pain and jaundice,—a steady pain, not one that comes and goes. I do not think I should consider it.

A STUDENT: How about the onset? Is there anything characteristic of cancer or ulcer?

DR. CABOT: Certainly yes. In the vast majority of cases the onset of ulcer is a very long, slow history, lasting for a period of years with spaces of good health. The course of cancer is usually one of a few months, perfectly steady progress, getting worse and worse. So that just the history alone will set you right in most cases.

A STUDENT: What is the type of symptoms?

DR. CABOT: There are no characteristic symptoms; we see pain, vomiting or nausea. But we get all the gastric symptoms in any gastric disease. It is the relation of symptoms to meals, their coming and going, their duration and their mode of relief that make the differentiation. Why he had such a poor appetite I do not know. He ought to have had a good appetite.

A STUDENT: Is there still a possibility of cancer?

DR. CABOT: Yes. I am glad you brought that up. The surgical observation may have been wrong, especially if they were working pretty fast. But I think it is unlikely.

A STUDENT: Does ulcer affect the sphincter often?

DR. CABOT: Quite often it is in the pyloric sphincter itself.

A STUDENT: And cancer?

DR. CABOT: Yes. Both in the same place often.

A STUDENT: The leucocyte count is rather high, is it not?

DR. CABOT: If he had, as I believe he did, a peritonitis at the end it is not high.

You remember they found at operation that the pancreas was the base of the ulcer. In other words there had been perforation before they got there, and it is possible that that earlier high leucocyte count was the result of a perforation not recognized at the time.

DR. MALLORY: The high leucocyte count was on the day that those X-rays were taken.

A STUDENT: There was some retention towards the end. How do you explain it?

DR. CABOT: You get it with cancer or ulcer.

A STUDENT: I always thought retention from an ulcer was caused by scarring at the pylorus.

DR. CABOT: It may be caused by spasm perfectly well without any scarring. It is a common symptom of ulcer. A contracted cicatrix is not common, rather rare.

A STUDENT: Do you think it possible that this man may not have had his worst symptoms spontaneously, but as a result of barium?

DR. CABOT: I wish there were an X-ray man here. The best I know is yes, it seems to be quite possible that the barium did it. I do not know enough to say that. I am speaking off-hand.

DR. MALLORY: Another possibility is, I think, in accordance with all the evidence that this perforation occurred at the time of or within a very few hours after the X-ray examination. If you consider the method of palpation during fluoroscopic examinations, with a wooden lever vigorously wielded, it seems very probable that this man was the victim of too much hospital study.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Duodenal ulcer with perforation.
Duodenal fistula.
Secondary hemorrhage.
Acute general peritonitis.

DR. RICHARD C. CABOT'S DIAGNOSIS

Duodenal ulcer with perforation and hemorrhage.
Localized peritonitis.
Arteriosclerosis.
Hypertrophy and dilatation of the heart.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesion.*

Duodenal ulcer with perforation and hemorrhage.

2. *Secondary or terminal lesions.*

Peritonitis, localized.
Bronchopneumonia.

DR. MALLORY: In our first abdominal incision we came down upon half a liter of bloody fluid, which was in a localized pocket in the upper right hand quadrant, bounded by the liver, the stomach and the transverse colon. Sponging that away exposed immediately the posterior wall of the duodenum. The anterior wall had entirely ulcerated away. In fact it would be better to say that we came down directly upon the pancreas. The base of the ulcer was formed by the head of the pancreas. It was entirely in the second portion of the duodenum, nothing in the pylorus, nothing in the stomach, except about a liter of partially changed blood.

There was a localized peritonitis in the upper half of the abdominal cavity, but well walled off by the omentum. It did not extend throughout the cavity. Very often in these large ulcers in which there is a great deal of terminal hemor-

rhage it is possible to find macroscopically an eroded blood vessel in the base of the ulcer of sufficient size to account for the hemorrhage. We were not able to do that however in this case, but the terminal hemorrhage must have been very considerable. We do not know how much had leaked out through the abdominal wound, although the report suggests that at least a liter had. The small intestine was filled with partially clotted blood.

His heart showed only slight hypertrophy. It weighed 370 grams and in general was normal in appearance. The coronary arteries were in good condition, and there were no scars in the myocardium.

There was no great evidence of arteriosclerosis anywhere. The aorta was smooth. The kidneys were negative.

There was a terminal bronchopneumonia.

DR. CABOT: I take it you realize that peptic ulcer, gastric or duodenal, is usually a fairly mild disease. Here we see one of the rare cases where it kills. A considerable number of the teachers in the Harvard Medical School have had it for years. It is so common among doctors that most of them do not stop working, do not get operated upon, and change their diet very little. We see of course the worst cases here, but we must realize that it is very common and on the whole a very mild disease.

CASE 13412

PRECORDIAL PAIN WITH AND WITHOUT EXERTION

MEDICAL DEPARTMENT

A Nova Scotian carpenter fifty-eight years old was brought to the Emergency Ward March 11 complaining of sudden attacks of severe pain over the heart. He was placed immediately upon the dangerous list. During the history taking he died.

In his youth he was in bed two months with an attack of acute rheumatic fever. His daughter said that for twenty years he had been subject to attacks of difficulty in getting his breath lasting from two hours to two days. The patient said he had never been dyspneic until the Christmas before admission. He had always done vigorous work. At no time had he had orthopnea or edema. He denied venereal disease.

One morning early in December, three months before admission, he was seized while walking with sudden severe pain over his heart radiating down his left arm so that he could not use it. The pain lasted only a very few minutes after he reached home. Not long after this while having all his upper teeth pulled he had another similar attack of sudden severe precordial pain lasting a few minutes and suddenly stopping. A week before admission he had another attack. After this he had similar attacks daily, at first one or two, then more frequent until the night

before admission he had fourteen, only temporarily relieved by ten pearls of amyl nitrite. He used five dozen pearls in the five days before admission. The last attack was not relieved by amyl nitrite. His physician gave a quarter of a grain of morphia and sent him to the Emergency Ward. There he had an attack not entirely relieved by nitroglycerin.

Clinical examination showed a very blanched, cyanotic man under the influence of morphia but able to rouse and answer questions intelligently. He lay flat on his back and was obviously not decompensated. The respiration was slow. The location of the apex impulse of the heart is not recorded. By percussion the left border of dullness was 10.5 centimeters from midsternum, 3 centimeters outside the mid-clavicular line, right border normal, supracardiac dullness 3.5 centimeters. The action was absolutely irregular; too rapid and the sounds too faint and too poor in quality to make it possible to note murmurs over the noise of respiration. The pulse was just perceptible at the wrist, 118 in the Emergency Ward. The systolic blood pressure was 90 in the Emergency Ward. In the medical ward no sound was heard, but the beats came through at 60. At the apex the pulse rate was so rapid that a count was not attempted. Electrocardiogram showed auricular fibrillation, rate 80 to 100, intraventricular block, probably right bundle branch. The pupils were pin-point, but contracted a little more to light. The abdomen and lungs were normal. The rest of the routine examination was not completed.

The patient was placed on the dangerous list at 12.25 p. m. He had a severe attack of precordial pain just as digalin was about to be given intravenously. He was given 10 cubic centimeters. At 1.40 the day of admission he died.

DISCUSSION

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY

1. The death of the patient during the history taking is a thing which we ought to remember as a possibility. A man comes in very sick. We are on duty for the afternoon or evening. We get busy getting in all the data for the visiting physician. We are working pretty hard. Sometimes, as in this case, the patient is not there when the visiting man comes next day. In order to get all our data in during the afternoon or evening we may overwork the patient, though the results are not often serious.

2. That he had always done vigorous work is an important fact. If he had been having as much dyspnea as his daughter suggested it does not seem as though he could have done vigorous work.

3. Weakness in the arm often goes along with pain in cases of angina.

4. The fact that the last attack was not re-

lieved by amyl nitrite is very important. Sometimes even half a grain of morphia will not stop this pain.

That is a very strong history of one thing and not of anything else. We may be entirely wrong, but nevertheless it means one thing.

A STUDENT: A blocked coronary?

DR. CABOT: Yes; cardiac infarction is what that history says. We may be wholly wrong, but nevertheless that is what the history says.

NOTES ON THE PHYSICAL EXAMINATION

"He lay flat on his back and was obviously not decompensated." You see he is dying, but not of decompensation. That generally means, if it is his heart, as it seems to be, that thing that I have named.

DIFFERENTIAL DIAGNOSIS

I think he died, as I said before, of cardiac infarction due to a blocked coronary. The only point worth discussing is the question of angina pectoris, which I think can be and should be differentiated from the disease which I believe this is. Angina pectoris is a common disease, much commoner than the disease which I believe this to be. Angina ought to be produced by exertion or emotion only, ought to stop with rest, ought to stop with nitrites, ought to last but a few minutes. There are very few cases on record that lead to death. There are a good many cases called fatal angina such as the death of Thomas Arnold, the father of Matthew Arnold. But we have no good reason to suppose that they were angina. They were continuous pain, not affected by drugs or exertion. This disease, if it is the disease I suppose it to be, may have gone so far as to produce a cardiac aneurysm, that is a thinning and bulging of the heart wall, and that may have gone so far as to break, although it occurs under very different circumstances. I do not believe one can tell ruptured heart from cardiac infarction. They all go together, cardiac infarction, thinning of the heart wall, aneurysm and sometimes rupture. Most cases do not rupture. I have seen it a number of times this year. It is amazing how common it seems to be, as contrasted with twenty or thirty years ago, when no one ever made the diagnosis.

A STUDENT: Should you say that he had angina pectoris at first?

DR. CABOT: If I had seen only these attacks and nothing else, yes. That is, I think the one disease can go into the other.

A STUDENT: Can you prove a case of angina post mortem?

DR. CABOT: No. You may find changes in the coronaries or in the aorta, or you may find no changes. We do not know the etiology of angina.

A STUDENT: Isn't that history enough? He had precordial pain lasting two or three months.

DR. CABOT: Yes, I think it is very much like angina.

A STUDENT: Is it typical to have so many attacks of pain in infarction?

DR. CABOT: Not typical. But it is certainly not at all like angina to have so many when the patient is in the hospital. It looks to me as if there was a little bit of exertion and then the attack occurred.

A STUDENT: Do you think the coronaries are arteriosclerotic?

DR. CABOT: Judging by his age I should think they were.

A STUDENT: Do you think the attacks were definitely angina?

DR. CABOT: I think you cannot look at the early attacks except in the light of the latter ones. I believe we know what the pathology was at the end. If I am right it is something that we do not call angina.

A STUDENT: Up to the time that his doctor was unable to give him relief and he was sent into the hospital wasn't the history purely that of angina?

DR. CABOT: I think it may have been. I do not think our history is very good. At the time he had his teeth pulled they say the pain lasted a few minutes and suddenly stopped. If it occurred during rest, as I suppose it did, that is not like angina, even though it stopped.

A STUDENT: I should like to ask Dr. Mallory if there is any pathologic relation between angina and coronary thrombosis.

DR. MALLORY: From the pathologic point of view I should consider angina as a symptom and coronary occlusion as a disease. Angina is purely a symptom, and we may or may not get suggestive evidence of it post mortem. If we find arteriosclerosis in the coronaries we guess that he has had an angina, but as often as not he has been symptom free.

DR. CABOT: If you look in the chapter on angina in my book on the heart you will find the entire experience in this hospital about this; it tells exactly how many had angina and showed coronary change and how many did not. I think you will arrive at the conclusion that you cannot say anything about its cause.

A STUDENT: When a man dies with angina pectoris what does he die of?

DR. CABOT: Coronary sclerosis, blocked coronary, and cardiac infarction.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Angina pectoris.
Auricular fibrillation.
Old rheumatic heart.
Coronary occlusion.

DR. RICHARD C. CABOT'S DIAGNOSIS

Coronary sclerosis; blocked coronary.
Cardiac infarction.

ANATOMIC DIAGNOSES

1. Primary fatal lesions.

Syphilitic heart disease, aortic regurgitation.

Syphilitic aortitis.
Chronic myocarditis (syphilis?)
Coronary occlusion.

2. Secondary or terminal lesion.

Chronic passive congestion.

DR. MALLORY: I am sorry the history was so inadequate, for although the man denied venereal disease he unquestionably died of syphilis. He showed a marked degree of luetic involvement of the aorta, chiefly localized to a short zone extending from the aortic valve upward for a distance of 2 centimeters, then stopping abruptly. In this area there were numerous stellate, puckered scars at the base of each of which a definite interruption of the yellow elastic coat of aorta could be made out, even in gross. Microscopic examination confirmed the luetic nature of the process. The remainder of the aorta showed a considerable degree of ordinary arteriosclerosis, without luetic involvement. The luetic process had extended down and involved the valves themselves. Ordinarily in attempting to determine the etiology of lesions of the aortic valve we note particularly the relations of the valve cusps at the corners, where they come most closely together. Normally the two cusps meet and become adherent to the aorta at a single point. In rheumatic infections the cusps become interadherent for a varying distance out from the attachment to the aorta. In a syphilitic process on the contrary ulceration occurs at the mutual point of attachment of the aorta and hence the cusps appear to arise from two separate points. That was quite evident in this case. The luetic process had also involved the mouths of the coronary arteries. The left was found without difficulty, but the right could only be located by tracing the artery back from below. A small gummatous process had completely blocked off the mouth of the coronary at its entrance to the aorta. Beyond that point the artery was patent and free from thrombosis. There was no large infarction of the myocardium, but numerous small fibrous scars were present, particularly in the interventricular septum. The histology of these suggests a luetic process rather than the ordinary fibroses following circulatory block.

The heart was considerably hypertrophied, weighing 480 grams. The left ventricle however was not greatly dilated, so that it is fair to argue that the degree of aortic regurgitation could never have been very marked.

The remaining organs showed generalized chronic passive congestion, but were otherwise not remarkable.

DR. CABOT: I think we have to say that is a clean miss in diagnosis. If we were trying to save ourselves we should say he has a blocked coronary, but it was blocked in the arch of the aorta, at the mouth of the artery itself.

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VANDERBILT HALL

AN epoch in medical education will be made tomorrow with the dedication of Vanderbilt Hall, the new dormitory of the Medical School of Harvard University. In the afternoon, Dr. George E. Vincent, President of the Rockefeller Foundation, will deliver the dedicatory address, after which the invited guests will have an opportunity to inspect the dormitory, and in the evening the Alumni Association will entertain at dinner in Bowditch Hall, under the presidency of Dr. Philemon E. Truesdale.

The first steps towards securing a dormitory for the students at the Harvard Medical School was taken some three years ago by the Alumni Association of the School, and a considerable sum of money was raised for the purpose among its members and among those friends who realized the importance of such a project for the health and well-being of the students. The end sought was finally achieved through the generosity of Mr. Harold Vanderbilt who made up the balance of the necessary sum, and for whom the dormitory is named.

Though credit is given to the Alumni Association for launching the project, and to Mr. Vanderbilt for completing it, it must not be forgotten that the finished dormitory is the con-

crete realization of the vision and a monument to the tireless efforts of Dr. Elliott P. Joslin, President of the Alumni Association at the time the idea was conceived. As a result of his enthusiasm and zeal and the unceasing energy of Dr. Francis M. Rackemann, Secretary of the Dormitory Committee of the Association, the building now stands ready to be dedicated tomorrow to the service of those who would train themselves to the service of others.

Although it has been called a dormitory this building will serve other purposes than the simple housing of students, for through its gymnasium (Mr. Vanderbilt's original gift), its great dining hall and its common living rooms it becomes the open club and social centre of the School.

Fittingly enough the names and memories of former graduates who have achieved success in the service of mankind will be perpetuated within its walls; thus the dining hall is named for Bowditch, the living room for Mixter, and over forty of the students' suites for other alumni who have brought credit to their School. The Student Room itself is named for Charles Best, the medical student and co-discoverer of insulin.

May Vanderbilt Hall serve its purpose well; may the names of those of generations gone, enshrined within it, serve as perpetual inspiration to those generations who will come to use it.

THE MEASLES PROBLEM

EPIDEMIOLOGISTS are very generally paying attention to the diminution of the preventable mortality incident to measles.

The aphorism that measles attacks without distinction but kills with discrimination is being quoted by writers on this subject.

This discrimination is based on the mortality found among children afflicted with measles of three years or younger. Older children have a comparatively low mortality although deaths are by no means rare and usually due to complications. The experience of the first American troops from the middle west of the United States during the World War demonstrates the fact that certain epidemics have an impressive mortality rate in ages above childhood.

Even with recovery this disease imposes economic burdens and disruption of normal living routine of no mean importance.

The application of immunizing methods began as far back as 1759 by Francis Home but there are no records of his methods.

Dr. A. K. Chalmers according to W. S. C. Copeman (*Proceedings of the Royal Society of Medicine*, Vol. XX, No. 10, Page 1610) some forty years ago appointed a vaccinator who blistered measles convalescents and vaccinated contacts with the serum. Although he claimed good results his ideas were not adopted.

Anderson and Goldberger in 1916 demon-

strated immunity in monkeys after the injection of blood from a measles case. Park and Zingher at about this same time secured favorable results. Richardson and Corvnor of Boston in 1918, Degkwitz in 1919, and later Miller and Smith of Harrogate, confirmed the experiments of others.

These and many others facts are set forth in the Proceedings of the Society of Medicine referred to above.

Enough has been reported to indicate that the problem is now largely a public health responsibility and methods must be devised for the use of immunization on as large a scale as possible. Thoroughly well-trained practitioners may be expected to use the proper agents to advantage but health departments should set about the collection of immunizing serum and be prepared to have field agents who can go to places as soon as an epidemic starts, apply the appropriate treatment and teach practitioners how to collect and apply the serum taken from convalescent cases.

The public should be taught that measles is far more serious disease than is generally considered and that immunization may be brought about more generally through the use of serum than will be secured through natural processes.

Thus far immunization may not be available for all victims because of the difficulty of securing enough serum but a large proportion of those who would come in the age range of greater mortality can be saved.

How long immunity may exist is not known at present but if a child can be carried safely through the early years of life, even if the disease is contracted later he may not run the greater risk. Enough is known already to warrant the widest possible use of this treatment and further studies may bring about as definite control of measles as now exists with smallpox.

Perhaps obstructive tactics may inhibit the activities of health departments but there are many who appreciate and accept scientific medicine.

AN IMPORTANT MEETING

For fifteen years the subject of Tuberculosis has not been given a prominent place in the meetings of the Suffolk District Medical Society. This omission is probably due to the feeling that the therapeutic and public health problems have been given very largely in the hands of specialists and organizations.

It must be recognized, however, that tuberculosis is often associated with many disorders and presents complicating problems in the routine of private practice and public health administration.

Its lowered death rate and diminishing incidence has perhaps led to some indifference with respect to its importance for although not the captain of the hosts of death it still plays a

great part in the attacks made by disease on the human race. Every practitioner may to advantage keep himself well-informed about the various manifestations of this disease.

Through the influence of Dr. John B. Hawes, 2nd, the Suffolk District Medical Society will devote the evening of October 26 to tuberculosis, and Dr. Allen Krause will speak on "Tuberculosis and the Medical Profession." Dr. Krause is an inspiring speaker and he will present the subject in a way to stimulate a sense of responsibility in the minds of physicians with respect to this disease.

Some pessimists have felt that the last word has been spoken on tuberculosis but when Drs. Baldwin and LoGrasso spoke before the Trudeau Society the hall was crowded.

Drs. Chadwick, Bowditch, O'Donnell, Remick, Christian, Pratt and Lord have been asked to participate in the discussion.

The importance of the subject, the ability of the reader of the paper, and the standing of those who will discuss the paper warrant the expectation of a very interesting meeting.

We hope that the doctors outside of Boston will appreciate the work which is done by the Suffolk District for all who are interested are welcome at their meetings.

Since Dr. Krause and the others are willing to devote an evening to this subject everyone who may find time to attend should show due courtesy to these gentlemen by their presence.

REFERENCES TO THE COUNCIL MEETING OF THE MASSACHUSETTS MEDICAL SOCIETY

A GOODLY number of Councillors met at the Boston Medical Library at noon, Wednesday, October 5, to greet the new President, Dr. John M. Birnie.

The meeting was promptly called to order and after the reading of the records the scheduled program was followed and the business dispatched quickly without much debate until the last article in the call for the meeting was reached.

The Committee of Arrangements made no report probably because of uncertainties with respect to the necessity of changing the date due to conflict with the time of the meeting of the American Medical Association in 1928, and the possibility of holding the Annual Meeting away from Boston.

Dr. F. H. Washburn, President of the Worcester District Medical Society, submitted a cordial invitation from the Worcester District to hold the next Annual Meeting in Worcester stating that the last meeting in that city was in 1851. The invitation was promptly and enthusiastically accepted, and Dr. A. W. Marsh of Worcester was appointed chairman of the local committee to act in conjunction with the Committee of Arrangements, with authority to select

his associates. Subsequent to this action, the Committee of Arrangements was given authority to fix the date of the next Annual Meeting of the Society either before or after the meeting of the American Medical Association.

A committee consisting of Drs. J. W. Bartol of Boston, W. L. Burrage of Brookline, H. G. Stetson of Greenfield, H. W. Van Allen of Springfield, and Henry Jackson, Jr., of Boston, was appointed to report a revision of the By-Laws.

Dr. Thomas J. O'Brien, delegate to the Maine Medical Association, reported attendance at the Annual Meeting of this Society and expressed appreciation of the cordiality and abundant hospitality enjoyed by his associate and himself.

Dr. H. W. Van Allen of Springfield was appointed delegate to the Vermont Medical Society.

The President referred to the loss sustained by the Council in the death of Dr. H. E. Buffum and, upon nomination of Dr. J. A. McLean to fill the vacancy, the Council voted approval. Another vacancy created by the resignation of Dr. H. D. Arnold, was filled by the election of Dr. S. F. Curran. Because of his loyalty to the State, Dr. Arnold has been willing to give up his official membership in the Society that he may serve on the Board of Registration in Medicine. Dr. Arnold's resignation also created a vacancy in the Committee of Nine in charge of the JOURNAL and Dr. F. T. Lord was elected to fill this position.

Although the President explained that the Society would need more money if its activities were to be extended and adequate rebates were to be given to the District Societies, the dues for the coming year were fixed at eight dollars.

Under new and incidental business several important matters were presented for discussion. The first was introduced by Dr. J. W. Bartol who explained the necessity of having an executive assistant to the President. Many duties now being rendered by the officers of the Society without compensation would be performed by this assistant. His motion was seconded by Dr. E. H. Stevens of Cambridge, Dr. C. E. Mongan of Somerville, Dr. W. L. Burrage, and Dr. T. J. O'Brien. The last speaker referred especially to the necessity of having an influential deputy of the Society who could be called upon to represent it at various functions and before legislative committees in particular. The Council expressed most cordial approval of the plan, when Dr. O'Brien reported that Dr. James S. Stone, ex-President of the Society, had consented to serve in this capacity. We feel sure that it is the unanimous opinion of the Council that there is no one so well qualified to fill this important position. The vote gives to the President power to appoint a salaried assistant.

The Committee of Nine, under the chairmanship of Dr. Homer Gage of Worcester, was given authority to represent the Society in such exercises as might be appropriate in recognition of the one hundredth anniversary of the founding of the BOSTON MEDICAL AND SURGICAL JOURNAL.

Since certain complications requiring careful study exist with reference to liability insurance carried by members of the Society, it was suggested by the President that a committee consisting of Drs. A. G. Rice of Springfield, A. H. Crosbie of Boston, and C. A. Sparrow of Worcester be appointed to consider the subject and make a report through the JOURNAL presenting pertinent recommendations.

The last and most actively debated subject was introduced by a member of the Plymouth District Society, who brought forward a motion which, if carried, would have created a new standing Committee of the Society. His contention was based on the abuses incident to misplaced charity work in clinics and hospitals. Although there is, apparently, no difference of opinion with respect to the existence of these abuses, there were expressions of doubt about the possibility of accomplishing much good through action by the Society. A majority finally voted, however, to pass an amended motion leaving the matter in the hands of a committee composed of a representative from each of the District Societies. This committee will study the subject and report its conclusions.

The conduct of the meeting was admirable, and much business was speedily transacted.

This superficial and fragmentary statement will, we hope, lead to a careful perusal of the official record of the Proceedings to be published when prepared by the Secretary.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

OHLER, W. R., S.B., M.D. Harvard Medical School 1914, Instructor in Medicine at Harvard Medical School, Assistant Visiting Physician at the Boston City Hospital. His subject is: "What Can Be Learned from Urinary Examinations." Page 593. Address: 270 Commonwealth Ave., Boston.

SMITH, GEORGE GILBERT, A.B., M.D. Harvard Medical School 1908, F.A.C.S., Urologist at the Massachusetts General Hospital, Surgeon to the Huntington Memorial Hospital, Consultant in Urology at the Palmer Memorial Hospital. His subject is: "The Diagnosis of Renal and Ureteral Lesions from the Viewpoint of the Genito-Urinary Surgeon." Page 597. Address: 6 Commonwealth Ave., Boston.

BUGBEE, HENRY G., M.D. Columbia University

College of Physicians and Surgeons 1903, F.A.C.S., Attending Urologist to St. Luke's, Woman's, Lawrence and Lying-In Hospitals, New York, Consulting Urologist to Mountain-side Hospital, Montclair, N. J., Muhlenberg Hospital, Plainfield, N. J., and Vassar Brothers' Hospital, Poughkeepsie, N. Y. His subject is: "The Clinical Value of Kidney Functional Tests." Page 611. Address: 40 E. 41 St., New York, N. Y.

CHRISTIAN, HENRY A., A.B., A.M., LL.D., M.D. Johns Hopkins 1900, Hersey Professor of the Theory and Practice of Physics at Harvard University, Physician-in-Chief at the Peter Bent Brigham Hospital. His subject is: "Diuretics, Their Utility and Limitations." Page 614. Address: Peter Bent Brigham Hospital, Boston.

MISCELLANY

THE OCHSNER FOUNDATION FOR CLINICAL RESEARCH

IN order to promote Clinical Research the American College of Surgeons has been developing a department of Clinical Research for investigation of efficient methods of diagnosis and treatment of diseases and injuries for which no specific methods have been approved.

A pamphlet recently distributed by the American College of Surgeons explains the association of Dr. Albert J. Ochsner's name with this Foundation in the following words:

"Dr. Albert J. Ochsner, one of the most devoted Fellows of the College, was deeply interested and an enthusiastic believer in the results to be accomplished by the research work outlined. In view of this fact and because, in practical, scientific results the Clinical Research Department of the American College of Surgeons will prove a suitable memorial to the eminent surgeon who devoted his life to scientific service in behalf of mankind, it was the decision of the committee in charge to devote the funds donated by friends of Dr. Ochsner to the support of the work under consideration.

In behalf of the Clinical Research Department, individual Fellows of the College have already contributed \$100,000 to the Ochsner Foundation fund, the income from which is being used in support of the initial work necessary for the full carrying out of the plan. Very soon the income from not less than one million dollars will be necessitated.

After careful consideration the committee believes that the direct benefit that will accrue to humanity warrants our request for financial support. The Fellows of the College will continue to produce noteworthy results, and will also contribute liberally to the foundation fund; and in this the coöperation of many members of the medical profession will be generously giv-

en. The American College of Surgeons accepts the responsibility of the custody of all funds contributed and insures the income being devoted to the work herein referred to.

We invite your interest, your influence with others and your own generous subscription."

At the meeting of the American College of Surgeons in Washington, May 18, 1927, the following resolution was submitted:

"Whereas the American College of Surgeons has established 'The Ochsner Foundation for Clinical Research' in affectionate and appropriate memory of Albert John Ochsner, of Chicago, Illinois, a distinguished Fellow of the College, one of its founders, its President and Treasurer during several years, and

Whereas the practical and continued services rendered by the Department to suffering humanity and the opportunities afforded for study and increased skill and efficiency in medical and surgical practice will, we believe, commend the Ochsner Foundation to the generous consideration of the American public,

Therefore Be It Resolved, that the plan should now be brought to the attention of the entire public, and

Be It Further Resolved, that the Board of Regents of the American College of Surgeons, in formal meeting assembled, endorses the proposed plan of establishing a nation-wide interest in the undertaking, and bespeaks for it the interest and support, not only of the medical profession, but of all citizens, and

Be It Further Resolved, that the Board of Regents endorses the Ochsner Foundation Committee, acting for and in the name of the American College of Surgeons,

And invests in the aforesaid Committee authority to pursue such plan as it may perfect for the raising of moneys toward the Foundation.

On motion duly made by Dr. Charles H. Mayo and seconded by Dr. Besley the resolution was adopted."

The Ochsner Foundation Committee consists of the following named persons: William J. Mayo, M.D., Chairman; Angus Hibbard, Vice-Chairman; Charles S. Bacon, M.D., Carl Beck, M.D., Frederic A. Besley, M.D., Truman W. Brophy, M.D., Herman N. Bundesen, M.D., Rush C. Butler, Walter E. Carr, W. W. Chipman, M.D., George W. Crile, M.D., Bowman C. Crowell, M.D., George W. Dixon, E. I. Erickson, Evan Evans, W. A. Evans, B.D., Ethan Allen Gray, M.D., Carl Hedblom, M.D., Charles E. Kahlke, M.D., Allen B. Kanavel, M.D., Philip H. Kreuscher, M.D., Franklin H. Martin, M.D., George A. Mason, Charles H. Mayo, M.D., T. B. Munroe, O. E. Nadeau, M.D., Rudolph Oden, M.D., Daniel A. Orth, M.D., Nelson M. Percy, M.D., S. R. Pietrowicz, M.D., Lorenzo T. Potter, M.D., George M. Reynolds, Lessing Rosen-

thal, Walter Dill Scott, LL.D., Erwin R. Schmidt, M.D., Henry Schmitz, M.D., John C. Shaffer, Kellogg Speed, M.D., Frank L. Stone, E. P. Strandberg, Meyer J. Sturm, Robert J. Thorne, John Milton Trainer, Melvin A. Traylor.

The committee has sent an appeal for contributions with the hope that an endowment fund of one million dollars can be raised for the purposes of the Ochsner Foundation.

Further information may be acquired by writing to Angus Hibbard, 40 East Erie St., Chicago.

TUFTS COLLEGE MEDICAL SCHOOL APPOINTMENTS 1927-1928

- Dr. A. Warren Stearns, Dean and Associate Professor of Neurology.
Dr. Timothy J. Murphy raised to Associate Professor of Pulmonary Diseases and Climatology.
Dr. Julius Loman, Teaching Assistant in Neurology.
Dr. Arthur Berk, Teaching Assistant in Neurology.
Dr. Francis P. McCarthy, Teaching Assistant in Dermatology.
Dr. Myron A. Stranmer, Teaching Assistant in Orthopedics.
Dr. Ruth Weissman, Teaching Assistant in Pediatrics.
Dr. Edward O'Brien, Teaching Assistant in Pediatrics.
Dr. Letitia Adams, Instructor in Surgery.
Dr. Grace E. Rochford, Instructor in Surgery.
Dr. Blanche L. Atwood, Instructor in Surgery.
Dr. Bernard Devine, Instructor in Surgery.
Dr. Joseph A. Meledy, Instructor in Theory and Practice of Medicine.
Dr. Horace K. Boutwell, Instructor in Pulmonary Diseases and Climatology.
Dr. Richard H. Houghton, Instructor in Pulmonary Diseases and Climatology.
Dr. Mark H. Joreess, Teaching Assistant in Pulmonary Diseases and Climatology.
Dr. Nathan Sidel, Teaching Assistant in Pulmonary Diseases and Climatology.

INFANTILE PARALYSIS IN NEW YORK

ONLY four new cases of poliomyelitis, or infantile paralysis, were reported at the Health Department from the five boroughs during the twenty-four hours ending at 10 A. M. October 1, and fear of an epidemic is unjustified, it was said by Health Commissioner Harris.

It is believed at the Health Department that the peak of the disease was reached during the week ended Sept. 3, when there were sixty-six new cases. The number dropped the following week to thirty-nine, but increased to fifty-three during the week ended Sept. 17. There were thirty-seven new cases in the week ended Sept. 21. Thus far there have been thirty-nine new

cases, in the week ending October 1, Dr. Harris said:

"In many parts of the country physicians and health officials have noted an increase in the prevalence of acute anterior poliomyelitis during the last two months, as compared with the corresponding period in 1926. While sufficient time has not elapsed to justify the assertion that a biennial occurrence of an increase in the number of cases is probably a characteristic of the disease, such biennial recurrence has nevertheless been noted since 1921.

"In April, 1920, and in March and May, 1921, no cases were reported to the Department of Health. Our records show the increase in incidence becomes distinctly noticeable ordinarily in July and reaches a maximum in August and September, occasionally continuing at a high level in October.

"The year 1924 is notable in that there was a persistence of acute anterior poliomyelitis, which appeared rather typical, having been most prevalent in September, October and November. The disease, as judged by past experience, subsides slowly, reaching its lowest level as a rule in December and almost invariably in January. Incidentally, it is to be noted that in our records the disease was not recognized in 1909 or in the preceding years.

"The total number of cases to Sept. 20 last was 363. While one cannot speak lightly of an incidence of 363 cases in a year, still, one must bear in mind the ratio of the cases per 100,000 children under 15 years of age. This is the arbitrary age group division, because the disease, while most prevalent among children of this age group, also occurs among adults.

"There were 174 cases among 597,100 children under 5 years of age, or, in other words, a rate of twenty-nine cases per 100,000. As there were 108 cases in the group of from 5 to 9 years, the rate is twenty-two cases per 100,000.

"This statement is not aimed to set forth the clinical types of the cases that are encountered in successive years nor to present a medical survey of the subject. It is merely to present to physicians and public health officials some data by means of which they can get a birdseye view of the situation."

FIFTH NEW ENGLAND HEALTH INSTITUTE

PROVIDENCE, R. I., SEPTEMBER 27-30, 1927

FOUR hundred and one public health workers were registered at the Fifth New England Health Institute held at the State House and the State College of Education, Providence, Rhode Island, from September 27-30. Dr. Byron U. Richards, Rhode Island State Commissioner of Health, was Director of the Institute and Dr. Marion A. Gleason was Chair-

man of the Program Committee. The Institute was held under the auspices of the State Departments of Health of the New England States, cooperating with the United States Public Health Service, the Yale and Harvard Schools of Public Health, the Departments of Biology and Public Health of Massachusetts Institute of Technology and Simmons College.

The following courses were scheduled:

1. *Public Health Administration.* 8 Lectures. Stanley H. Osborn, M.D., Commissioner, Connecticut Department of Health.
2. *Preventable Diseases.* 8 Lectures. Clarence L. Seamman, M.D., Director, Division of Communicable Diseases, Massachusetts Department of Public Health.
3. *Sanitation and Engineering.* 6 Lectures. L. M. Fisher, Sanitary Engineer, U. S. Public Health Service, Providence, R. I.
4. *Tuberculosis.* 7 Lectures. Frank Kierman, President, N. E. Conference on Tuberculosis, Boston, Mass.
5. *Veneral Diseases.* 6 Lectures. George G. Smith, M.D., Boston, Mass.
6. *Child Hygiene.* 6 Lectures. A. Elizabeth Ingraham, M.D., Director, Bureau of Child Hygiene, Hartford, Conn.
7. *Public Health Nursing.* 8 Lectures. Edith L. Soule, R.N., Director, Division Public Health Nursing, Augusta, Me.
8. *Social Work.* 4 Lectures. Helen Powers, State Director, Mothers' Aid, Providence, R. I.
9. *Mental Hygiene.* 6 Lectures. Paul J. Ewerhardt, M.D., Providence, R. I.
10. *Industrial Hygiene.* 6 Lectures. Luther D. Burlingame, Providence, R. I.
11. *Foods, Food Control and Nutrition.* 6 Lectures. Charles Duncan, M.D., Secretary, New Hampshire State Board of Health.
12. *Health Education.* 6 Lectures. Charles F. Dalton, M.D., Secretary, Vermont Department of Public Health.

Miss Bernice W. Billings, R.N., Executive Secretary of the Boston Tuberculosis Association, presided over the opening session at which the speakers were Miss Mary Gardner, R.N., Director, Visiting Nursing Association, Providence, Rhode Island, who spoke on "Tuberculosis Nursing Program for a City," and Mrs. Theresa Anderson, R.N., State Supervising Nurse, Maine Public Health Association, Augusta, Maine, who spoke on "Tuberculosis Nursing Program for a Rural Community." Miss Harriet Leek, Director of the Visiting Nursing Association of New Haven, Connecticut, had as her subject "The Public Health Nurse as a Health Teacher."

Surgeon C. E. Waller spoke on the "Montreal Typhoid Fever Situation in Relation to Milk." Dr. Waller was of the group of representatives of the United States Public Health Service which went to Montreal and made an

exhaustive study of the typhoid epidemic of last winter and spring. Surgeon Waller stated that while it could not be definitely said just what the origin of the epidemic was their studies had demonstrated the likelihood that the failure to pasteurize the entire milk supply of the City seemed the most plausible explanation.

Mr. L. M. Fisher, sanitary engineer in the U. S. P. H. Service, stationed at Providence, spoke (Tuesday) on "Shellfish sanitation." He gave much in various lines concerning the scarcity of shellfish. He noted that the pollution of the waters, where there was not direct action of factory wastes, but rather the sewage of municipalities, lowered the content of oxygen and diminished animal and other life dependent on this. Much of his paper was devoted to the sketching of disease outbreaks supposed to be related to polluted shellfish. In the face of the diminishing production of grounds suitable for the propagation of unpolluted shellfish, the fact was brought forward by the speaker that in clear water the molluscs can cleanse themselves of the pollution and become marketable. This is particularly true of the oyster, and a considerable industry has sprung up in reclaiming oysters originally from polluted waters, but laid for a sufficient time in clear waters. The relaying of clams is a more serious problem on account of their burrowing habits and the mortality due to breakages in handling. The speaker urged the appropriation of funds for studies and investigation, whereby to reclaim not only the beds, but any shellfish originally in polluted waters. Incidentally Mr. Fisher noted that there had not been a natural set of oysters in Narragansett Bay for ten years.

Dr. Howard A. Streeter, health officer of Manchester, N. H., discussed "Sanitary Food Inspection," (on Tuesday morning), outlining the needs of the various situations that confront the food inspector in the city and in the country. He emphasized the importance of food selling within stores, with a minimum of dust and opportunity for contamination, and spoke rather discouragingly of the open-air markets, whose goods are likely to be no fresher than those of stores, and whose sanitary care may be questionable. He called attention to the fact that pure foods are not always assured by the laws and regulations. Noting a specific example, he said that butter without coloring matter, would not be saleable, and the law allows its sale, but oleomargarine if colored is absolutely forbidden for sale, whether the package be marked or not. For the conclusion of a helpful paper Dr. Streeter sketched the varying fundamental laws of the N. E. states. In Maine the Commissioner of Agriculture is the enforcing officer and in New Hampshire, the food laws are state legal enactments while the sanitary regulations come from the local boards of health. In Vermont there is a state law on pure foods and drugs,

the enforcement of the purity and sanitation being effected by local boards. In Massachusetts the local boards pass regulations backed by the State Department of Public Health. Connecticut has no general state sanitary code, practically the whole regulation being by local boards, and in Rhode Island a commission is in charge of the general state regulations. In all the New England states there are certain sanitary standards for foods, while the United States government has regulations governing foods in inter-state operations.

Willis E. Chandler, Executive Secretary of the Rhode Island Tuberculosis Association, presided at the joint session of the Tuberculosis Section and the Section on Public Health Nursing on Tuesday afternoon at which Professor C. E. Turner of the Department of Biology and Public Health of the Massachusetts Institute of Technology was the principal speaker.

Professor Turner spoke on "Health Education." He stressed the importance of articulating the subject of health education into the entire school curriculum. He described the complementary character of physical training and health education which are sometimes considered to be antagonistic. He outlined the plan of health education recently prepared under his direction for the City of Cleveland. The plan emphasizes training and instruction in health habits from the kindergarten through the high school. It is being put into effect beginning with this school term. A special Health Education Director has been employed by the School Department to supervise the operation of the new project.

Miss Anna W. Johnson, Educational Secretary of the Massachusetts Tuberculosis League, discussed Professor Turner's paper. Miss Johnson pointed out that the health education program for schools differs essentially from the special campaigns for safety, fire prevention, and similar special drives, in that it is an effort to weave into the entire school life the actual practice of sound health habits. Newton and Malden in Massachusetts are two outstanding cities in which health education methods, differing in type but having the same objective and the same results, are being successfully carried on.

Miss Johnson stated that the school physician and the school nurse are the natural promoters of a school health education program but that the principal work of training in health habits must be carried on by the class teacher. The health of the teacher herself and sound health habits are of great importance because the children are quick to detect any variation between the teaching of good health habits and the failure to practice them by the preceptress.

In the light of the wide-spread practice of furnishing milk for school children, both for underpar children and normal children, at the

luncheon period, Miss Johnson pointed out that it was a matter of supreme importance that the source of the milk supply and the quality of milk should be constantly watched.

Mr. John Ritchie of Malden, Mass., former health commissioner of the city of Boston, called attention to the almost entire absence of other than text-book health education in the public schools, instancing the schools of Malden and Newton, Mass., as pioneers in a most important line of practical health work.

The Tuberculosis Section was continued on Wednesday morning with Dr. Edward J. Rogers, Superintendent, Vermont Sanatorium, Pittsford, Vermont, as Chairman.

What has been done in New Hampshire through the community clinics, Dr. Robert B. Kerr, executive secretary of the N. H. Tuberculosis Association, set forth in an extremely helpful manner in the morning session on Wednesday. He emphasized the need of public health education and the formation of a public opinion strong enough to support rational health movements. As an educational feature the clinics are remarkably strong. Much of the paper was devoted to discussions of a technique that has proved successful in a state very largely rural in population, this population being spread over territory, some of which is practically impassable in winter. He noted that the work of the N. H. clinics is intended to be diagnostic in character, but if treatment is really necessary the association will finance it. Important is the follow-up, which is cared for by the association, and it has also a consultation service.

Dr. John F. O'Brien, Superintendent of the Seaside Sanatorium, Niantic, Connecticut, spoke on "The Care and Treatment of Non-Pulmonary Tuberculosis." Dr. O'Brien's paper was illustrated by motion pictures showing the treatment of bone and joint tuberculosis by heliotherapy.

Dr. Harry Lee Barnes, Superintendent of the Rhode Island State Sanatorium at Wallum Lake, described the methods by which the members of the patient's family are induced to co-operate with the officials of the Sanatorium in assisting in the successful sojourn of the patient at the Institute. Samples of the letters sent to prospective patients, relatives of patients, and parents of children who are patients at the Sanatorium were read.

At the invitation of the Trustees of the State Sanatorium at Wallum Lake, one hundred persons attending the Tuberculosis Section of the Institute were entertained at luncheon on Wednesday at the Sanatorium after which Dr. Harry Lee Barnes, Superintendent, gave a lecture on artificial pneumothorax and Dr. R. J. Rosenblatt gave a demonstration of the procedure of this method in tuberculosis.

A most interesting paper was that of Miss

Georgie B. Collins of Malden, Mass., on "Planning the School Health Education Program." (Wednesday morning) Miss Collins was a student under Professor Turner at the M. I. T., and was selected by him to undertake the experiment of practical health education in public schools. A trial was made for a year in a school in Somerville, Mass., but the next year Malden called for Miss Collins, and since that time, some four years ago, has steadily extended the program of health teaching.

Noting the extent and importance of the general subject, Miss Collins confined her discussion to the public school, and within this restricted matter, took up only the approach to the introduction of health work. The many difficulties in the way were outlined, insufficient appropriations, the enormous number and variety of subjects handed over to the class room teacher, the inertia of some administrations and the over energy of some who seek to have their subjects taken up by the schools. Miss Collins spoke of the tact required to gain the child, who first by rewards and later through appreciation becomes the principal factor in its own health education, in fact, under this program, the practical custodian of its own health. The approach to the teacher must be a proper one, for teachers have much thrust upon them which they are expected to develop themselves, and they are naturally wary about being imposed upon. When however, the idea of a practical health education is "sold" to them, they become most ardent workers. And of course it is foolish to try to proceed without the confidence and assistance of the school administration. Incidentally Miss Collins spoke of the appreciation of the school authorities in Malden, which has made the experiment in that city so successful.

Mr. Herbert W. Moses, Director of Personnel Work of the Edison Electric Illuminating Company of Boston, spoke on Thursday morning on "Industrial Health." He stated that of the 3,500 employees of the Boston Edison Company 3% are constantly absent from sickness. The loss to the Company in 1926 from sickness alone was 29,000 working days. Sickness, much more than industrial accidents, is the cause of loss of time from work among the employees of this Company. The loss of time from accidents in the same year was 4,600 days.

On Thursday and Friday afternoons six lectures dealing with sex problems and the control of venereal disease were given. Mr. Ray H. Everett, Editor of the American Journal of Social Hygiene, spoke on "What Social Hygiene Has Accomplished." Dr. Randal Hoyt of New York told of the advances in the treatment of neurosyphilis, and Dr. Austin Cheever of Boston described the ideal syphilis clinic. On Friday afternoon Dr. J. Edwards Kerney of Providence, Director of the Division of Social

Hygiene of the Rhode Island Board of Health, read a paper on "Advances in the Treatment of Gonorrhoea." Mr. Newell W. Edson, of the American Social Hygiene Association, spoke on "Sex Social Problems of Today," and Raymond S. Patterson, Ph.D., Chief of the Bureau of Venereal Diseases, New Jersey Board of Health, described the methods employed in New Jersey to clean up the foci from which venereal diseases are being disseminated. In New Jersey, the doctors are requested to report to the State Board of Health not only the case of venereal disease, but also the name of the person from whom the disease was contracted. Last year some 900 sources were so reported. The Board of Health then informs the Health Officer of the locality in which this person lives that such and such an individual is alleged to be infectious, but does not give the name of the informant. The local Health Officer then investigates in a cautious manner, and if the circumstances appear to bear out the information, the infectious individual is forced to undergo treatment.

The lectures herein summarized were only a portion of those read at the Institute. Many interesting topics were discussed by competent speakers, and frequently there was considerable discussion. On Thursday evening an Institute dinner was held at the Hotel Biltmore, at which Dr. George Bigelow was a speaker. Numerous trips to industrial plants, sanitary plants and welfare stations were arranged for members of the Institute. All persons interested in the subjects presented were invited to attend.

RECENT DEATHS

DUGGAN—DR. JOHN THOMAS DUGGAN, city physician for the last six years, and former Mayor of Worcester, died at his home in that city September 5, 1927, at the age of 72.

He was a native of Worcester, a graduate of the College of the Holy Cross in the class of 1880 and of Bellevue Hospital Medical College three years later. He served four three-year terms on the School Committee and was chairman of the United States Board of Pension Examiners for the Worcester district. In 1906 and 1907 he was Mayor of Worcester and for the past six years served as city physician. He joined the State medical society in 1886. He was Vice-President of Worcester District 1911-1912, and President of that District 1912-1913. He was a member of the staff of St. Vincent's Hospital.

He is survived by his wife, who was Nellie J. Glast-rick of New York, and by eight children.

DWINELL—DR. WILLIAM GROUT DWINELL, of Pawtucket, R. I., died in Providence, September 22, 1927, following a long illness, aged 49.

He was a native of St. Johnsbury, Vt. where he was born on June 9, 1878. He attended St. Johnsbury Academy and later the Malden High School, where he prepared for Harvard Medical School. He took his M.D. there in 1901 and settled in Pawtucket, with an office in Providence. In the World War he was a major in the Medical Corps. He wrote an article on epidemic pneumonia, published in the *American Journal of the Medical Sciences*, during the influenza

epidemic. During the past five years he had carried on a biochemical laboratory in Providence.

HARNEY—DR. ROBERT EDWIN HARNEY, a pediatrician of Dorchester, died at his home there, October 3, 1927, aged 47.

He was a graduate of Tufts College Medical School in 1914, joined the Massachusetts Medical Society in 1923 and was a member of Robert E. Fulton Council, K. of C., and of St. Peter's Court, M. C. O. F.

He is survived by his widow, who was Eleanor E. Carney.

HEBBARD—DR. ELLERY COLA HEBBARD, a retired member of the Massachusetts Medical Society, died at Boston, October 4, 1927, aged 80.

He was a graduate of Bellevue Hospital Medical College in 1870 and settled in Boston in 1874, joining the State society. He was a member of the American Association for the Advancement of Science, a 33rd degree Mason and one of the oldest practicing physicians in Boston.

He is survived by his wife, Ida M. Hebbard, who was one of the prime movers in woman suffrage in the early days, and also president of the Housekeepers' League.

KNOWLES—DR. EDWARD A. KNOWLES died September 18, 1927, after an illness of two days of bronchopneumonia (primary) acute dilatation of heart. He was born in Somerville, February 2, 1881. As a result of his war service in France he had a chronic pharyngolaryngitis due to gas poisoning and a nerve condition due to shell shock.

Dr. Knowles was a graduate of Tufts Medical College, class of 1912, cum laude.

LITTLEWOOD—DR. THOMAS LITTLEWOOD, of Pittsfield, died suddenly of heart disease while working on his car in his garage, September 13, 1927.

He was born in England September 30, 1880, graduated from the Worcester Tech, and after taking an M.D. at Dartmouth Medical School in 1907 he was assistant superintendent at the Gardner State Colony, Massachusetts, joining the State medical society in 1909. He moved to Pittsfield in 1915 and had practiced there since. During the World War he served with an ambulance company in France, attaining the rank of captain. He was a Fellow of the American Medical Association and a member of Hope Lodge of Masons of Gardner, and a member of the staff of the House of Mercy Hospital.

He is survived by his widow, two brothers and a sister.

OSBORNE—DR. CAROLINE AMELIA OSBORNE, an assistant in the Baldwinville Hospital, formerly of Worcester, died at Gardner, September 17, 1927, aged 61.

She was born in East Templeton, a daughter of Dwight C. and Maria J. (Howe) Osborne, and had been a resident of Worcester for the past 35 years. Death occurred after an illness of only two days. An abscess in her throat, discovered two days before her death, resulted in pneumonia, from which she died.

Dr. Osborne was a graduate of Wilbraham Academy and received her training as a nurse at the Worcester City Hospital. She received her medical degree from the Woman's Medical College of Pennsylvania, Philadelphia, in 1899, and then spent the next six years as superintendent of nurses at the Worcester Memorial Hospital. Later she received her degree of master of arts and of doctor of philosophy from Clark University. She practiced medicine for nine years in Worcester, and then became assistant physician at the Hospital Cottages for Children, Baldwinville, a position she held until her death.

She was a member of the Worcester District Medical Society, the American Medical Association and the

American Association for the Advancement of Science. She also had an active interest in the Worcester Association of Nurses, and was a member of the Wesley Methodist Church.

She is survived by two sisters in Worcester, Miss Lucy A. Osborne, a member of the faculty at the Girls' Trade School, and Miss Henrietta J. Osborne, an artist.

POWELL—DR. MAUD AGATHA POWELL, a member of the Massachusetts Medical Society since 1908, died at her home in Laconia, N. H., August 2, 1927, at the age of 53.

She was a graduate of the Woman's Medical College of Pennsylvania in 1906, started practice in Boston in 1908, moved to Laconia the next year and had practiced roentgenology there since.

SPARKS—The death has been reported of Dr. CLARENCE IRVING SPARKS, of Easthampton, by gas poisoning, September 15, 1927.

He was a graduate of Baltimore Medical College in the class of 1898 and had been associate medical examiner of the second district of Hampshire County for 14 years when he resigned in 1925.

He is survived by his widow.

WHITTEN—DR. GEORGE EDWIN WHITTEN, a Fellow of the Massachusetts Medical Society since 1891, died at his home in Haverhill, September 23, 1927, aged 68 years, after a long illness.

He was born in Parsonsfield, Me., August 10, 1859, his grandfather having been a general in the Revolution. He studied medicine at Bowdoin, changed to Dartmouth and received his M.D. there in the class of 1888. At first he practiced in Lawrence, in New York City and in New Haven, Conn., settling in Haverhill in 1896. He was a 33rd degree Mason.

In 1893 he married Miss Carrie Patch, who survives him.

OBITUARY

AMASA ELLIOT PAINE, M.D.

DR. AMASA ELLIOT PAINE, long time medical examiner of the Plymouth District, a veteran of the Civil War, died at his home in Brockton, September 30, 1927, aged 83. The son of Capt. Amasa and Susannah Freeman Paine he was born at Truro November 19, 1843. After a preparatory school education at Truro Academy, he entered Harvard Medical School in 1862. He left the school almost immediately, however, to enlist in the union army. He was at first assigned to E company, 43d Massachusetts infantry, but was subsequently transferred to the regimental hospital, where he acted as assistant to the head surgeon. Discharged in 1863, he returned to Harvard, where he remained until June, 1864, when he once more enlisted. He was appointed a medical cadet in the regular army and was assigned to the Mt. Pleasant Hospital at Washington, D. C. While on duty at Washington he studied at Georgetown University medical school, being graduated there in 1865. He was then promoted to be assistant surgeon of the 104th regiment of colored volunteers. He was finally discharged from the army in 1866.

Dr. Paine returned to Cape Cod, beginning practice at Wellfleet. In May, 1867, he married

Miss Lucie W. Ritter, daughter of Frederick W. Ritter of Washington, D. C.

In September, 1867, Dr. Paine moved to Brockton, then known as North Bridgewater, a town of only 7000 population. In 1877 he was appointed by the Governor to the office of medical examiner for the first Plymouth district, a position he held for 49 years, resigning last spring.

Dr. Paine was prominently identified with the Massachusetts Medico-Legal Society which he served as treasurer, and of the Plymouth District of the Massachusetts Medical Society, being president in the years 1891-1892. A councillor of the Parent Society, he was constant in his attendance at the meetings of that body until his retirement in 1920.

When the Brockton Hospital Association was formed Dr. Paine was one of its most active sponsors. He served as a member of the executive committee of the hospital and for several years was one of its vice-presidents. He was chairman of the medical board for years and chief of the consulting staff for many more years. He was a member of the Brockton Commercial Club, Massasoit lodge, I. O. O. F., the Loyal Legion, and Fletcher Webster post, G. A. R.

He is survived by his wife, Mrs. Lucie W. Paine, and two daughters, Mrs. Charles W. Fairbanks, formerly of Whitman and now of Mattapan, and Miss Charlotte H. Paine of Brockton.

MASSACHUSETTS MORTALITY STATISTICS: 1926

Washington, D. C., September 29, 1927. - The Department of Commerce announces that the 1926 death rate for Massachusetts was 1,254 per 100,000 population as compared with 1,248 in 1925. The principal increases in rates in 1926 were from diseases of the heart (from 249 to 259 per 100,000 population), influenza (from 15 to 20), nephritis (from 85 to 89), whooping cough (from 7 to 10), and measles (from 8 to 10).

A decrease in 1926 was shown in the death rate from diphtheria (from 8 in 1925 to 6 per 100,000 population).

| Cause of death | Deaths in Massachusetts | | | |
|--|-------------------------|--------|---------------------------------------|---------|
| | Number | | Rate per 100,000 estimated population | |
| | 1926 | 1925 | 1926 | 1925 |
| All causes 1/ | 52,638 | 51,721 | 1,254.2 | 1,248.0 |
| Typhoid and paratyphoid fever | 59 | 73 | 1.4 | 1.8 |
| Malaria | 2 | 3 | 2/ | 0.1 |
| Smallpox | - | - | - | - |
| Measles | 401 | 349 | 9.6 | 8.4 |
| Scarlet fever | 125 | 116 | 3.0 | 2.8 |
| Whooping cough | 412 | 279 | 9.8 | 6.7 |
| Diphtheria | 250 | 334 | 6.0 | 8.1 |
| Influenza | 850 | 604 | 20.3 | 14.6 |
| Dysentery | 12 | 16 | 0.3 | 0.4 |
| Erysipelas | 100 | 127 | 2.4 | 3.1 |
| Lethargic encephalitis | 76 | 97 | 1.8 | 2.3 |
| Meningococcus meningitis | 59 | 51 | 1.4 | 1.2 |
| Tuberculosis (all forms) | 3,503 | 3,441 | 83.5 | 83.0 |
| Of the respiratory system | 2,976 | 2,910 | 70.9 | 70.2 |
| Of the meninges, central nervous system | 239 | 251 | 5.7 | 6.1 |
| Other forms | 288 | 280 | 6.9 | 6.6 |
| Syphilis 3/ | 461 | 470 | 11.0 | 11.3 |
| Cancer and other malignant tumors | 5,313 | 5,172 | 126.6 | 124.8 |
| Rheumatism | 128 | 129 | 3.0 | 3.1 |
| Pellagra | 11 | 10 | 0.3 | 0.2 |
| Diabetes mellitus | 844 | 808 | 20.1 | 19.5 |
| Meningitis (nonepidemic) | 123 | 141 | 2.9 | 3.4 |
| Cerebral hemorrhage and softening | 4,092 | 4,057 | 97.5 | 97.9 |
| Paralysis without specified cause | 97 | 110 | 2.3 | 2.7 |
| Diseases of the heart | 10,865 | 10,324 | 258.9 | 249.1 |
| Diseases of the arteries, atheroma, aneurysm, etc. | 2,040 | 1,956 | 48.5 | 47.2 |
| Bronchitis | 314 | 346 | 7.5 | 8.3 |
| Pneumonia (all forms) | 4,906 | 4,884 | 116.9 | 117.9 |

MASSACHUSETTS MORTALITY STATISTICS (Continued)

Deaths in Massachusetts

| Cause of death | Number | | Rate per 100,000 estimated population | |
|--|--------|-------|--|-------|
| | 1926 | 1925 | 1926 | 1925 |
| Respiratory diseases other than bronchitis and pneumonia (all forms) | 320 | 277 | 7.6 | 6.7 |
| Diarrhea and enteritis (total) | 944 | 904 | 22.5 | 21.8 |
| Diarrhea and enteritis (under 2 years) | 812 | 773 | 19.3 | 18.7 |
| Diarrhea and enteritis (2 years and over) | 132 | 131 | 3.1 | 3.2 |
| Appendicitis and typhlitis | 530 | 552 | 12.6 | 13.3 |
| Hernia, intestinal obstruction | 437 | 431 | 10.4 | 10.4 |
| Cirrhosis of the liver | 238 | 228 | 5.7 | 5.5 |
| Nephritis | 3,713 | 3,541 | 82.5 | 85.4 |
| Puerperal septicemia | 181 | 167 | 4.3 | 4.0 |
| Puerperal causes other than puerperal septicemia | 355 | 378 | 8.5 | 9.1 |
| Congenital malformations and diseases of early infancy | 3,164 | 3,271 | 75.4 | 78.9 |
| Suicide | 465 | 455 | 11.1 | 11.0 |
| Homicide | 84 | 111 | 2.0 | 2.7 |
| Accidental and unspecified external causes (total) | 2,731 | 2,902 | 65.1 | 70.0 |
| Burns (conflagration excepted) | 212 | 218 | 5.1 | 5.3 |
| Accidental drowning | 224 | 271 | 5.3 | 6.5 |
| Accidental shooting | 27 | 28 | 0.6 | 0.7 |
| Accidental falls | 721 | 655 | 17.2 | 15.8 |
| Mine accidents | - | - | - | - |
| Machinery accidents | 63 | 66 | 1.5 | 1.6 |
| Railroad accidents | 122 | 107 | 2.9 | 2.6 |
| Collision with automobile | 9 | 14 | 0.2 | 0.3 |
| Other railroad accidents | 113 | 93 | 2.7 | 2.2 |
| Street-car accidents | 57 | 58 | 1.4 | 1.4 |
| Collision with automobile | 8 | 16 | 0.2 | 0.4 |
| Other street-car accidents | 49 | 42 | 1.2 | 1.0 |
| Automobile accidents (excluding collision with railroad and street-cars) | 682 | 729 | 16.2 | 17.6 |
| Injuries by vehicles other than railroad cars, street-cars, and automobiles 4/ | 60 | 86 | 1.4 | 2.1 |
| Excessive heat (burns excepted) | 35 | 65 | 0.8 | 1.6 |
| Other external causes | 528 | 619 | 12.6 | 14.9 |
| All other defined causes | 4,342 | 4,496 | 103.5 | 108.5 |
| Unknown or ill-defined causes | 91 | 110 | 2.2 | 2.7 |

1/ Exclusive of stillbirths.

2/ Less than one-tenth of 1 per 100,000 population.

3/ Includes tabes dorsalis (locomotor ataxia) and general paralysis of the insane.

4/ Includes airplane, balloon, and motorcycle accidents.

CONNECTICUT DEPARTMENT OF HEALTH

MORBIDITY REPORT FOR THE WEEK ENDING
SEPTEMBER 17, 1927

| | | | |
|--------------------------------|----|----------------|----|
| Diphtheria | 14 | Scarlet fever | 16 |
| Last week | 22 | Last week | 13 |
| Diphtheria bacilli carriers | 4 | Measles | 4 |
| Typhoid fever | 5 | Last week | 8 |
| Last week | 6 | Whooping cough | 52 |
| | | Last week | 44 |

| | | | |
|------------------------|----|----------------------|----|
| Bronchopneumonia | 5 | Poliomyelitis | 19 |
| Chickenpox | 12 | Septic sore throat | 2 |
| Encephalitis, epidemic | 1 | Tuberculosis, pulmo- | |
| German measles | 3 | nary | 33 |
| Influenza | 1 | Tuberculosis, other | |
| Mumps | 12 | forms | 7 |
| Paratyphoid fever | 2 | Gonorrhea | 21 |
| Pneumonia, lobar | 14 | Syphilis | 15 |

DISEASE INCIDENCE IN CONNECTICUT
WEEK ENDING SEPTEMBER 24

| | 1927 | | | | 1926 | | | | |
|------------------------|-------------------------|--------------------------|--------------------------|--------------------------|--|-------------------------|--------------------------|--------------------------|--------------------------|
| | Week ending September 3 | Week ending September 10 | Week ending September 17 | Week ending September 24 | Average cases reported for week corresponding to September 24 for past seven years | Week ending September 4 | Week ending September 11 | Week ending September 18 | Week ending September 25 |
| Actinomycosis | - | - | - | - | - | - | - | - | - |
| Anthrax | - | - | - | - | - | - | - | - | - |
| Botulism | - | - | - | - | - | - | - | - | - |
| Cerebrospinal Men. | - | - | - | 2 | 1 | 1 | 2 | 1 | 2 |
| Chickenpox | 11 | 8 | 12 | 2 | 7 | 2 | 2 | 3 | 6 |
| Conjunctivitis, infec. | - | - | - | - | - | - | - | - | - |
| Diphtheria | 8 | 22 | 14 | 17 | 30 | 9 | 8 | 8 | 10 |
| Dysentery, Amoebic | - | - | - | - | - | - | - | - | - |
| Dysentery, Bacillary | 1 | - | - | - | - | - | 1 | - | - |
| Encephalitis, Epid. | 1 | - | 1 | - | - | - | 2 | - | - |
| Favus | 1 | - | - | - | - | - | - | - | - |
| German Measles | - | - | 3 | 1 | - | 1 | 1 | 1 | 1 |
| Hookworm infection | - | - | - | - | - | - | - | - | - |
| Influenza | 2 | - | 1 | 1 | 1 | 1 | - | 2 | - |
| Leprosy | - | - | - | - | - | - | - | - | - |
| Malaria | 2 | - | - | - | 2 | 1 | - | 1 | 2 |
| Measles | 4 | 8 | 4 | 3 | 8 | 9 | 6 | 2 | 4 |
| Mumps | 2 | 6 | 12 | 7 | 2 | 1 | 1 | 3 | 3 |
| Paratyphoid Fever | 1 | 1 | 2 | - | - | 1 | 1 | - | - |
| Pneumonia (Broncho) | 17 | 7 | 5 | 9 | 8* | 7 | 4 | 11 | 9 |
| Pneumonia (Lobar) | 8 | 10 | 14 | 14 | 7 | 16 | 10 | 15 | 11 |
| Poliomyelitis | 19 | 11 | 19 | 12 | 5 | 1 | 3 | 2 | 2 |
| Scarlet Fever | 3 | 13 | 16 | 18 | 28 | 13 | 19 | 17 | 20 |
| Septic Sore Throat | - | 3 | 2 | 1 | - | 18 | 1 | 1 | 1 |
| Smallpox | - | - | - | - | - | - | - | - | - |
| Tetanus | 1 | 1 | - | - | - | - | - | - | - |
| Trachoma | - | - | - | - | - | - | - | - | - |
| Trichinosis | - | 2 | - | - | - | - | - | - | - |
| Tuberculosis (pul.) | 17 | 34 | 33 | 13 | 26 | 33 | 11 | 29 | 32 |
| Tuberculosis (o.f.) | 4 | 6 | 7 | 1 | 3 | 2 | - | 1 | 1 |
| Typhoid Fever | 3 | 6 | 5 | 9 | 14 | 8 | 9 | 8 | 10 |
| Typhus Fever | - | - | - | - | - | - | - | - | - |
| Whooping Cough | 32 | 44 | 52 | 38 | 40 | 36 | 23 | 17 | 25 |
| Gonorrhoea | 41 | 30 | 21 | 26 | 18 | 66 | 10 | 16 | 12 |
| Syphilis | 58 | 35 | 15 | 28 | 25 | 32 | 8 | 10 | 9 |

*Average for two years. Made reportable January 1, 1925. Remarks: No cases of cholera, Asiatic, glanders, plague, rabies in humans and yellow fever during the past seven years.

NOTICES

AN INVITATION FROM THE MASSACHUSETTS SAFETY COUNCIL

THE Massachusetts Safety Council has extended an invitation to the members of the Massachusetts Medical Society to attend a meeting of its Engineering Section, to be held at the Engineers Club, Arlington Street and Commonwealth Avenue, Boston, on Thursday eve-

ning, October 13, at 6:30 p. m., for the discussion of the "Prone Pressure Method of Resuscitation." The speakers will be Stephen E. Whiting, Liberty Mutual Insurance Company; "When Used." Sydney W. Ashe, General Electric Company, Pittsfield; "How Used." Dr. Samuel H. Littlefield, United Drug Company; "Why Used."

There will be a \$2 charge for the dinner. Reservations should be made in advance through

the Massachusetts Safety Council, 6 Beacon Street; Tel. Haymarket 2101.

Physicians not personally familiar with the Schaefer method of resuscitation, which has been generally adopted by police and fire departments and the public service corporations, will find this program of practical value.

REPORTS AND NOTICES OF MEETINGS

ANNUAL CONFERENCE OF THE NATIONAL COMMITTEE FOR THE PREVENTION OF BLINDNESS IN CO-OPERATION WITH ILLINOIS SOCIETY FOR THE PREVENTION OF BLINDNESS

OCTOBER 13-15, 1927, CHICAGO, ILLINOIS
HEADQUARTERS: HOTEL STEVENS

PROGRAM

THURSDAY, OCTOBER 13, 1927

9:30 A. M. *Relation of Venereal Diseases to Vision Impairment.*

Joint session with Illinois Social Hygiene League. Presiding: A. J. Chesley, M.D., Executive Officer, State Department of Health, St. Paul, Minn.

1. Bacterial Invasion of the Birth Canal; Its Menace to the Newborn. E. L. Cornell, M.D., Assistant Professor of Obstetrics, Northwestern University, Chicago, Ill.

2. The State's Function in Promoting Prophylaxis of the Newborn's Eyes. Don M. Griswold, M.D., Deputy Commissioner, State Department of Health, Lansing, Mich.

3. Syphilitic Eye Tragedies and Their Prevention. John Green, M.D., St. Louis, Mo.

4. Early Treatment in Relation to Eye Tragedies. W. L. Benedict, M.D., Mayo Clinic, Rochester, Minn.

Discussants: Olin West, M.D., Secretary, American Medical Association, Chicago, Ill.

Hugh O. Jones, M.D., Assistant Commissioner of Health, Chicago, Ill.

James Stewart, M.D., State Health Commissioner, Jefferson City, Mo.

William A. Evans, M.D., Health Editor, Chicago Tribune, Chicago, Ill.

William F. Snow, M.D., General Director, American Social Hygiene Association, New York City.

2 P. M. *Relation of Proper Lighting to Conservation of Vision.*

Joint session with Illuminating Engineering Society.

Presiding: E. C. Crittenden, Bureau of Standards, Washington, D. C., and Lewis H. Carris, Managing Director, National Committee for the Prevention of Blindness.

1. How We Learn to See and How We Learn Some Other Things. W. T. Bovie, Assistant Professor, Biophysics, Harvard University, Cambridge, Mass.

2. Intensity of Light and Speed of Vision, Studied with Special Reference to Industrial Situations. C. E. Feree and Gertrude Rand, Department of Psychology, Bryn Mawr College, Bryn Mawr, Pa.

3. Four Fundamental Factors in Seeing. P. W. Cobb and F. K. Moss, Lighting Research Laboratories, National Lamp Works of the General Electric Company, Cleveland, Ohio.

Discussion by Mr. Cravath, Illuminating Engineer, Chicago, Ill., and others.

7:30 P. M. *Illuminating Engineering Society Banquet*, Crystal Ballroom, Edgewater Beach Hotel.

Delegates of the National Committee for the Prevention of Blindness are invited to attend. Tickets, at \$3.00 each, may be obtained from the Secretary.

FRIDAY, OCTOBER 14

9:30 A. M. *Eye Accidents in Industry.*

Joint session with the National Safety Council.

Presiding: Walter G. King, President of the National Safety Council, Chicago, Ill.

1. Economic Aspect of Industrial Eye Injuries as Reflected in Workmen's Compensation Laws, in Compensation Payments, and in the "True" Cost of Eye Accidents. Stewart J. Owen, Jr., Safety Engineer, National Safety Council, Chicago, Ill.

2. Prevention of Industrial Eye Injuries Through Efficient Safety Devices and Through Methods of Goggle Distribution. Harry Guilbert, Director, Bureau of Safety and Welfare, Pullman Company, Pullman, Chicago, Ill.

3. The Effect and Control of Injurious Invisible Light Rays in Industrial Occupations from the Medical and Research Point of View. Charles Sheard, Ph.D., Section on Physics and Bio-physical Research, Mayo Clinic, Rochester, Minn.

4. The Effect and Control of Injurious Invisible Light Rays in Industrial Occupations from the Practical Shop Point of View. J. L. Benash, Consulting Engineer, Chicago, Ill.

12 M. *Luncheon Conference on Eye Hazards in Industrial Occupations.*

Presiding: Lewis H. Carris, Managing Director, National Committee for the Prevention of Blindness. Face to Face with the Blind Man.

2 P. M. *Necessity for Correcting Remediable Eye Defects in School Children.*

Joint program with the Illinois Society for the Prevention of Blindness.

Presiding: Harry S. Gradle, M.D., Chicago, Ill.

Address by William H. Wilder, M.D., Emeritus Professor of Ophthalmology, Rush Medical College, Chicago, Ill.

Discussion led by Walter Cornell, M.D., Philadelphia, Pa.

5:15 P. M. *Ophthalmology in Relation to Prevention of Blindness.*

Program arranged in coöperation with the Chicago Ophthalmological Society.

SATURDAY, OCTOBER 15

9:30 A. M. *Sight-Saving Classes—Their Role in Conservation of Vision.*

Presiding: Mrs. Winifred Hathaway, Associate Director, National Committee for the Prevention of Blindness.

1. The Oculist's Relation to the Sight-Saving Class. E. V. L. Brown, M.D., Professor of Ophthalmology, University of Chicago, Chicago, Ill.

2. Essential Teacher Training for Sight-Saving Class Work. Miss Estella Lawes, Director, Department for Sight-Saving, Cincinnati Public Schools, Cincinnati, Ohio.

3. The English Sight-Saving Class. Miss Olive Peck, Assistant Supervisor, Department for the Blind and Sight-Saving Classes, Cleveland, Ohio.

4. The Philosophy of the Sight-Saving Class. Lewis H. Carris, Managing Director, National Committee for the Prevention of Blindness.

Discussants: John B. Curtis, Supervisor, Department for the Blind, Chicago Public Schools, Chicago, Ill.

Miss Alice V. Burdge, Supervisor, Sight-Saving Classes, State Department of Education, Columbus, Ohio.

Miss Gladys E. Dunlop, Supervisor of Sight-Saving Classes, Detroit, Mich.

Mrs. Rena Cummings, Supervisor of Sight-Saving Classes, Seattle, Wash.

12 M. *Luncheon Conference on Sight-Saving Classes.*

Presiding: E. V. L. Brown, M.D., Professor of Ophthalmology, University of Chicago, Chicago, Ill.

1. Who Shall Be Admitted to Sight-Saving Classes?

Miss Gladys Dunlop, Supervisor of Sight-Saving Classes, Detroit, Mich.

2. What the Teacher Should Know About Eye Conditions. Mrs. Winifred Hathaway, Associate Director, National Committee for the Prevention of Blindness.

3. Cooperation of the School Oculist and the Teacher with the Private Ophthalmologist. B. Franklin Royer, M.D., Medical Director, National Committee for the Prevention of Blindness.

4. Training the Sight-Saving Teacher in the Classroom. Frank G. Bruner, Ph.D., Director of Special Schools, Chicago, Ill.

Adjournment for round table discussions at close of luncheon. Time and place to be announced.

2 P. M. *Trachoma as a Public Health Problem.*

Joint session with the Standing Committee on Conservation of Vision of the Conference of State and Provincial Health Authorities of North America.

Presiding: William H. Wilder, M.D., Emeritus Professor of Ophthalmology, Rush Medical College, Chicago, Ill.

1. Prevalence Among Appalachian Mountaineers and the Cooperation Plan of Relief Between United States Public Health Service and State Health Departments. Major Paul D. Mossman, Surgeon, United States Public Health Service, Rolla, Mo.

2. Minnesota's Plan of Using Indian Nurses. A. J. Chesley, M.D., Executive Officer, State Department of Health, St. Paul, Minn.

3. Wisconsin's Plan of Supplying Public Health Nurses. C. A. Harper, M.D., State Health Officer, Madison, Wis.

4. Trachoma in Palestine. Charles F. Wilinsky, M.D., Deputy Commissioner of Health, Boston, Mass. Discussants: W. F. Cogswell, M.D., Secretary, State Department of Public Health, Montana.

Don M. Griswold, M.D., Deputy Commissioner, State Department of Health, Lansing, Mich.

J. A. Stucky, M.D., Lexington, Ky.

Opportunity will be given for visits to headquarters of the Illinois Social Hygiene League and to the clinic administered by that organization.

Technique used in testing vision of pre-school children will be demonstrated each day by Mrs. Jessie Ross Royer, R.N., staff associate of the National Committee for the Prevention of Blindness, during the Conference at Stevens Hotel, Private Dining Room No. 2.

Opportunity to visit sight-saving classes in the Chicago schools will be given those interested.

CENSORS' MEETING

THE Censors of the Suffolk District Medical Society will meet for the examination of candidates at the Medical Library, No. 8 The Fenway, Thursday, November 3, 1927, at 4:00 o'clock.

Candidates should make personal application to the Secretary, and present their medical diploma at least one week before the examination.

ARTHUR H. CROSBIE, *Secretary*.
520 Commonwealth Avenue, Boston

MIDDLESEX NORTH DISTRICT MEDICAL SOCIETY

THE Middlesex North Medical Society will have its next regular meeting at the State Infirmary Tewksbury, Oct. 19, 1927, in conjunction with the other Medical Societies, Middlesex South, Essex North and South. A very interesting program has been arranged by the Hospital Staff.

T. A. S.

UNION HOSPITAL IN FALL RIVER

CLINICAL STAFF MEETING

THE Regular Monthly Clinical Staff Meeting will be held at the Stevens Clinic on Thursday, Oct. 20, 1927, at 8:15 P. M. All physicians interested are cordially invited.

M. N. TENNIS, M.D., *Secretary*.
Oct. 7, 1927.

ESSEX NORTH DISTRICT MEDICAL SOCIETY

THE 86th quarterly meeting of this Society will be held at State Infirmary, Tewksbury, Mass., (Telephone Lowell 160) upon invitation of Superintendent John H. Nichols, M.D., Wednesday, Oct. 19, at 1 P. M. sharp, and will be a joint meeting with these District Societies: Essex South, Middlesex East and Middlesex North. From 1 to 2 there will be ward visits to Men's Hospital, Women's Hospital, Children's Hospital, Tubercular Wards, and Mental Wards, starting from Administration Building.

2 P. M. sharp in the Chapel, clinics will be held by members of Hospital Staff as follows:

1. Welcome by G. Forrest Martin, M.D., of Lowell, Chairman of Board of Trustees.

2. Announcements by Superintendent.

3. Tuberculosis clinic by Jas. F. Lawlor, M.D.

4. Discussion opened by Sumner H. Remick, M.D., of Boston, Director Division of Tuberculosis, State Board of Health.

(a) Men's Hospital Clinic, Surgery, Cancer and Arthritis, by Arthur K. Drake, M.D.

(b) Heart Disease and Aneurism, by G. Herbert Cleary, M.D.

(c) Neurological Cases, by Wm. R. Izzo, M.D.

(d) Discussions opened by Hardy Phippen, M.D., of Salem.

6. Women's Hospital Clinic. Special cases by Geo. A. Peirce, M.D.

7. Congenital Syphilis, by Samuel A. Dibbons, M.D.

8. Talks with cases. "Mutism in Mental Cases," by Chas. L. Trickey, M.D., and Chas. J. Carden, M.D.

4 P. M. sharp: 1. Talk, Social Problems in Psychiatry, by A. Warren Stearns, M.D., of Boston, Dean of Tufts College Medical School (35 minutes).

2. Discussion opened by Francis W. Anthony, M.D., of Haverhill, Vice-Chairman Board of Trustees.

3. Remarks by Mr. Richard K. Conant of Boston, Commissioner of Public Welfare.

4. Remarks concerning the Massachusetts Medical Society by its President, John M. Birnie, M.D., of Springfield.

5 P. M. Luncheon.

All topics open to discussion.

The next meeting of the Censors will be held at Hotel Bartlett, 95 Main Street, Haverhill,

(Telephone 3430) Thursday, Nov. 3, at 2 P. M. sharp.

ADELBERT M. HUBBELL, M.D., *President.*

J. FORREST BURNHAM, M.D., *Secretary.*

567 Haverhill St., Lawrence, Mass.

Oct. 11, 1927.

THE ANNUAL MEETING OF THE AMERICAN SOCIETY OF TROPICAL MEDICINE

THE 23rd annual meeting of The American Society of Tropical Medicine will be held in Boston, Oct. 21 and 22, in the Amphitheatre Building E, Harvard Medical School.

Dr. George C. Shattuck, Harvard Medical School, Boston, is the president of the Society.

BENJAMIN SWARTZ, *Secretary.*

P. O. Box 131, Pennsylvania Avenue
Station, Washington, D. C.

ANNUAL MEETING OF THE N. E. SURGICAL SOCIETY

OFFICIAL REPORT

THE tenth annual meeting of the New England Surgical Society was held at Manchester, N. H. on September 30th and October 1st.

A large number of members were on hand at the Elliot Hospital Friday morning at nine o'clock to listen to an admirable "dry clinic" by the various New Hampshire members. A list of cases shown follows:

Dr. Elmer J. Brown, Manchester, N. H.; Dr. A. S. Merrill, Manchester, N. H.—1. Kidney and ureteral calculi. X-ray lantern slides.

Dr. James B. Woodman, Franklin, N. H.—1. Chronic intestinal obstruction. 2. Dermoid cyst.

Dr. Ezra Jones, Manchester, N. H.—1. Fracture tibial head with displacement semilunar cartilage.

Dr. Sam S. Dearborn, Nashua, N. H.—1. Dislocation of hip (with slides).

Dr. Thomas W. Luce, Portsmouth, N. H.—1. Massive fracture of skull. 2. Compound dislocation of shoulder.

Dr. David W. Parker, Manchester, N. H.—1. Closed drainage in empyema.

Dr. Geo. C. Wilkins, Manchester, N. H.—1. Carcinoma of cervix. Results of radium treatment.

Dr. James W. Jameson, Concord, N. H.—1. Acute pancreatitis following cholecystectomy. 2. Pyonephrosis. 3. Ureteral kink.

Dr. Geo. C. Wilkins, Manchester, N. H.—1. Tubercular cervical adenitis. Combined surgical and radium treatment. 2. Drainage in caesarian operation. 3. Case papillary cystadenoma of ovary. 3 years after operation.

Dr. David W. Parker, Manchester, N. H.—1. Prolapse of rectum in children. 2. Case of hypernephroma in an infant. 3. Osteomye-

litis lower jaw. 4. Combined gastric and duodenal ulcer. Operation.

Dr. Carleton R. Metcalfe, Concord, N. H.—

1. Case. Stabilization of foot.

This clinic was run off with great promptness and at eleven o'clock an adjournment was taken to the Operating Room where Dr. Wilkins did an appendectomy through an incision all his own and a pan hysterectomy and Dr. Parker did a sub-total Thyroidectomy and a new simple suture method for the relief of prolapse of the rectum in a child.

A delicious standing lunch was provided by the Hospital authorities. At two o'clock the Scientific program was begun at the Hotel Carpenter and continued until five.

At seven o'clock the members began to gather at the Manchester Country Club for a very pleasant social hour and at eight o'clock 73 members sat down to a most excellent dinner. After dinner the President, Daniel F. Jones of Boston, delivered his presidential address (to be published later) and then introduced the Hon. George H. Moses, United States Senator from New Hampshire who delighted the gathering with an interesting talk. Then followed an hour of story telling by various members, inspired doubtless by the delightful air of Manchester.

On Saturday the Scientific program was continued at the Carpenter, interrupted at noon by an excellent sit down lunch.

At the executive session after luncheon the following officers were elected for the ensuing year:

President, Robert B. Osgood, M.D., Boston.

Vice-President, George C. Wilkins, M.D., Manchester, N. H.

Secretary, John M. Birnie, M.D., Springfield, Mass.

Treasurer, Peer P. Johnson, M.D., Beverly, Mass.

Recorder, Walter G. Phippen, M.D., Salem, Mass.

The President announced that Dr. Cheever had been re-appointed to the Executive Committee for another term and that the next meeting would be held in New Haven, Conn.

The Papers of the afternoon were wisely limited to four so that the exercises concluded early enough for men to leave after the meeting. This avoided the gradual shrinking of the audience during the reading of papers—so often seen—when the last speaker nervously wonders whether he must perchance read to empty chairs.

Much thanks are due to Drs. Wilkins and Parker for arranging such a delightful meeting. Everything went off with precision and smoothness and all agreed that it was one of the best of the always good New England Surgical Society meetings.

WALTER G. PHIPPEN, M.D., *Secretary.*